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DISEASES OF THE LUNGS

DISEASES OF THE LU

DESIGNED TO BE A PRACTICAL PRESENTATION
THE SUBJECT FOR THE USE OF STUDENTS
AND PRACTITIONERS OF MEDICINE

BY

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PREFACE

THE original contract between the publishers and the autho a work of one volume which in approximately 1,000 pages s the entire field of diseases of the chest. As the preparation of proceeded it became apparent to the author that such an arrange prove unsatisfactory, since it would necessitate too great a cond make the work of practical value. Accordingly he prevailed up lishers to bring out the work in two volumes. This they were a ready to do since they had grown discouraged over the length of t consumed and were fearful lest the book might never be compl

Accordingly when the portion dealing with diseases of the arterial system had reached almost the size originally intended fo subject it was given to the public in 1903. Its reception was mo and far exceeded the author's expectation. Encouraged by the his first effort in that direction he took up the task of prepari volume with redoubled vigor. Unfortunately this one has also slowly and it is only after a lapse of three years that he is read it to the criticism of the reviewer and of such other readers as it with their perusal.

That this volume contains numerous defects the author is and he awaits with considerable apprehension the criticisms to upon the chapters devoted to pneumonia and pulmonary tuberc has endeavored, however, to present these as well as other su manner that will render them available to the student and pra medicine, since the book is not intended for the experienced inte knowledge may surpass that of the writer.

There is nothing novel in method and there is no wearisom of unfounded theories. The aim has been to be practical and hen diagnosis and treatment have received especially full considerati work proves of practical utility to the reader, the author will amply rewarded.

In conclusion he desires to express his thanks to all who h wise contributed to the preparation of the work. Thanks are due lar to his illustrator Dr. Milton W. Hall who has taken great pains in having the figures illustrate and not merely adorn the text. The photographs of pathological specimens are from preparations in the museums of the Rush Medical College and the Cook County Hospital. As with his former work the author is also deeply indebted to his wife for her aid in revising the manuscript and reading the proof.

ROBERT HALL BABCOCK.

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SECTION I

DISEASES OF THE BRONCHI

CHAPTER I

BRONCHITIS

Introductory.—Previous to the work of the immortal Laenne subject of diseases of the chest was enveloped in much obscurity. owing not so much to ignorance of pathology as to lack of satisfac of clinical differentiation of the various pulmonary, bronchial affections. When at length Laennec discovered auscultation, an alongside of percussion as a means of diagnosis, he and his succ enabled to distinguish most of the intrathoracic diseases recognize

Most of the questions which engaged attention in the beginning century have now become matters of ancient history. Neverthele remain and possess great practical importance, namely, what is the bronchitis, and in how far this can be utilized as a basis for termin the past many terms were employed in the attempt to classify va of the disease. Some were symptomatic, as in Laennec's Dry a while much more recently others have been proposed that rest on ical basis, e. g. Farrand's Congestive, Catarrhal and Inflammat classifications are not in keeping with modern conceptions and fa with the trend of present-day thought, which is to apply to disc that are based on their ætiology.

It is highly desirable, furthermore, to render this subject as possible. Accordingly Marfan's classification will be adopted in while at the same time is retained the very useful division into Chronic Bronchitis. Marfan distinguishes two varieties of bronc mation, based so far as our present knowledge permits upon thei Thus the bronchitis which occurs in infectious or specific disorder Specific; whereas that which results from extension from above or ical or mechanical irritation is called Nonspecific.

The adjectives Primary and Secondary are also sometimes appl chitis and in many ways are convenient. For instance the bronc depends in a certain measure at least upon chronic disorders of heart, kidneys or blood vessels is spoken of as Secondary. The these terms lies in the liability of confusing the so-called Primar bronchitis, or that following exposure or acute coryza, with the sp chitis seen in Measles, Pertussis, Influenza, etc. If this distinction is kept strictly in mind there can be no great objection to the employment of Primary and Secondary, and since they are sometimes highly convenient they will find occasional employment in the following pages.

Ætiology. Rôle Played by Bacteria.—It can now be definitely stated that micro-organisms are the immediate or exciting cause of bronchitis observed in diphtheria, influenza, measles, pertussis, pneumonia, etc. In some cases they reach the bronchi from without in consequence of direct infection from some individual suffering from the disease or through inhalation of germs floating in the dust of the atmosphere. In 220 cases of diphtheria, Councilman, Mallory and Pearce found bronchitis in most instances. In 42 the large bronchi were the seat of a fibrinous exudate. In influenza there is proof of the presence of the bacillus in the secretions of the throat and in the sputa; so that their detection here affords a reliable means of diagnosis of the nature of the infection.

Although the specific organisms of measles and whooping-cough have not yet been identified, still there can be no doubt that these diseases are due to some particular microbe to which the symptomatic bronchitis may also be attributed. Regarding pertussis it is interesting and worthy of note that Dr. D. J. Davis, a worker in the laboratory of Rush College under Dr. Ludwig Hektoen, while studying an epidemic in Chicago in 1905 among the foreign population of the West side, found the influenza bacillus in everyone of 22 cases examined. The organism was contained in the thick tenacious mucus raised after a paroxysm of coughing.

The bronchitis seen in enteric fever, in variola and the early stage of malaria, scarlatina, etc., is of hæmatogenous origin, either the germs themselves or their toxins being thus carried to the bronchial mucosa. In variola it is thought possible that the characteristic eruption may take place on the bronchial mucosa, the same as it does in the throat. Whatever may be the irritant in these diseases the bronchitis may justly be classed as specific. The same holds true of the bronchitis seen in fibrinous pneumonia and in cases of pneumococcus infection without demonstrable pneumonic exudate. As is now well known Fraenkel's diplococcus is always recognizable in the sputum of such cases. Other micro-organisms capable of exciting bronchial inflammation are Friedländer's pneumobacillus, the streptococcus pyogenes, staphylococcus pyogenes albus and aureus.

The action of these microbes is not confined to acute cases, but is responsible for numerous instances of chronic bronchitis. This is especially true of the influenza bacillus and of the pneumococcus, both of which are frequent findings in the sputum of chronic bronchitis. Thus I recall the case of a lady who had suffered for many months from an intractable cough with purulent expectoration and who was brought to me for diagnosis because of the fear that her symptoms might be due to tuberculosis. The lungs were resonant but exhibited throughout a multitude of fine and medium-sized mucous râles. Careful examination of the sputa failed to disclose tubercle bacilli but on the contrary numerous influenza bacilli and streptococci. In such a case the chronic bronchitis was probably an outgrowth of an acute inflammation.

Passing now from bronchitides of specific, or in other words (tionably bacterial origin, we come to the highly important query, play any rôle in the production of *nonspecific bronchitis,* to use classification? In other words, Is the discovery of micro-organis expectoration of those forms known as primary and secondary an discovery, or do they exert a distinct ætiological influence? This qu cult if not impossible of definite answer. It will probably never be determine by experimentation whether or not germs act as exciting nonspecific as well as specific forms of bronchitis, since it would sible to render human beings or animals wholly free from the p germs in their mouths and air-passages before subjecting them fluence of exposure and mechanical or chemical irritation of the mucosa. Hence we are compelled to conjecture concerning the eff presence of micro-organisms in connection with certain predisposi For my part I entertain the view that bacteria do play a most impc in the production of all cases of acute bronchial inflammation.

The bacterial flora normally present in the throat and respira including trachea and main bronchi, is rich and varied. At the sam organisms are harmless on account, mainly, of the fact that so lo mucous membrane is healthy, conditions for their growth are u As soon, however, as the protection furnished by the normal epi impaired, the germs take advantage of the opportunity thus affc begin to grow. In short, a culture medium has been provided in germs may flourish and multiply.

Such a favoring condition may be some irritant which destroys t of the epithelial covering of the mucosa in a circumscribed area, disturbance of the circulation in consequence of which the nutrit or the secretions become so altered in character as to prove a good s culture of the bacteria normally present; or the condition may l constitutional state which lessens tissue-resistance. Whatever it acts merely as a predisposing cause of the bronchial inflammation, germs themselves excite the inflammation by their active growth

Such a conception of the ætiology of bronchitis accords with o notions of acute disease and renders intelligible much that is oth scure. It is in harmony, moreover, with certain facts that are every-day observation. I refer to the "contagiousness" of some unimportant inflammations of the nose and throat. It is not at mon for one member of a family to display a coryza, or an ordinary as it is called, and before long it has spread through the entire This is not strange when we consider the intimate relation existir members of a family. The infection, for such it may be termed, from one to another through contact.

If such an infection or contagion is possible between members o there is nothing against the assumption that when a bronchitis foll in the head or a sore throat by extension, it is in reality through of the infective agent along the surface of the respiratory tract or the lymph channels. Indeed such seems to me the only rational e of such cases and to furnish a strong argument in favor of the bacterial origin of all cases of acute bronchitis of the class we have chosen to group under Marfan's term of Nonspecific.

Various investigators have endeavored to determine in how far microorganisms may be responsible for bronchial inflammations, independent of the acute infections. In the first place, it was necessary to ascertain whether or not the lower air-passages contain germs in health. The difficulties in the way of determining such a point are great and hence results have been conflicting. It is believed by some that the germs with which respired air is laden are deposited upon the mucosa of the nares and pharynx and that hence the air is practically sterile when it reaches the bronchi.

In support of this view is the observation that the expired air is free from germs, and the belief that the mucous membrane lining the nares possesses bactericidal properties. Moreover, Jundell is said to have examined the trachea of 43 persons during life by means of a specially devised instrument, and to have found it either sterile or to contain bacteria in limited numbers and for only a transitory period.

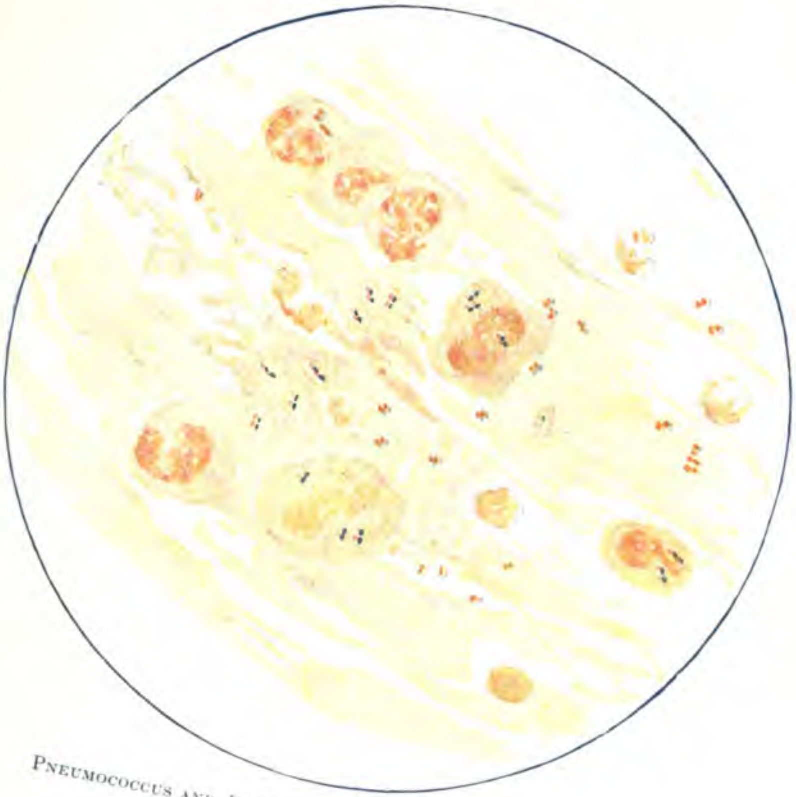
Others, on the contrary, have arrived at an opposite conclusion so that by some writers, West e. g., it is stated as an accepted fact that bacteria do normally exist in the trachea and main bronchi. Some even maintain that microorganisms exist in the bronchioles and alveoli (Dürck).

It is unfortunate that this point has not been definitely settled. Nevertheless, as I conceive it, it renders all the more valuable from an ætiological standpoint the discovery of germs in the lungs and bronchi in cases of bronchial and pulmonary inflammation, as well as in the sputa of such cases. Thus in numerous instances of bronchitis with fever, but with otherwise mild symptoms, R. Pfeiffer identified a peculiar kind of micrococcus which he designated Diplococcus Catarrhalis (Plate I.). It occurs in pairs, is somewhat larger than the staphylococcus and presents certain characters that make it resemble the gonococcus. Ghon and H. Pfeiffer are said to have confirmed his findings. Fraenkel, however, does not commit himself to any opinion concerning the value of their discoveries.

Other investigators have not discovered peculiar forms to which they attach definite specific properties but have recognized various well-known pathogenic organisms. Thus D. G. Ritchie carefully investigated the lungs of children dying of acute bronchitis and reported in every instance the presence of numerous germs, among which were streptococci, staphylococci, colon bacilli, influenza and diphtheria bacilli, the bacillus pyocyaneus and encapsulated bacilli. Diplococci of pneumonia and streptococci were the most common, however, and in the majority of cases a mixed infection was present. Joseph M. Patton of Chicago has reported 2 cases of bronchitis in which the symptoms, fever, loss of resonance and signs of bronchitis, were caused by streptococci and staphylococci discovered in the sputum. The bronchitis was cured by antiseptic inhalations.

Coming now to the consideration of the ætiology of *chronic bronchitis* we find difficulties in the way of accepting bacteria as the cause in every instance. It is very possible for a streptococcus bronchitis, e. g., to become subacute or

PLATE I



Pneumococcus and Diplococcus Catarrhalis in Sputum.

chronic, the same as may one due to the influenza bacillu
cases in which the influence of bacteria is doubtful or at
refer to those forms called congestive and catarrhal.

In such, the dividing line between passive hyperæmia
gestion of inflammation is not clearly drawn. In the hype
dry or results in scanty viscid mucus while in the catarrhal t
abundant but is still composed mainly of mucus. Bacteria
or are present in very small numbers. The underlying con
mechanical interference with the circulation.

There is a chronic bronchial catarrh, but symptoms of in
scarcely be said to exist. Are germs, Pfeiffer's coccus catarrhali
sible for the irritation and increased secretion of bronchial m
these be attributed to the congestion *per se?* For the present
must be left unanswered. It is very likely that in cases display
or muco-purulent expectoration micro-organisms are responsible
sistence and obstinacy. In other words, the germs prolong the c
do not actually produce them.

The attacks of acute bronchitis to which these cases are so prone
ever probably owing to the action of micro-organisms and the int
these may persist after the acuteness of the exacerbation has subsi
the form known as *fœtid* or *putrid bronchitis*, germs of course play
conspicuous rôle as will be shown in the proper place. On the whole
be assumed that germs exert more or less influence in all cases of chr
well as acute nonspecific bronchitis, but just how important an influenc
not be stated in the present state of our knowledge.

Conditions Predisposing to Bronchitis.—Extension.—In the m
ity of instances acute bronchitis follows a coryza or pharyngitis in consequ
of extension of the inflammatory process along the mucous membrane.
some cases the initial symptoms of the bronchitis are experienced so soon af
the "cold in the head" that both are thought to be the expression of a co
acting simultaneously or nearly so in both regions. In others the coryza pr
cedes the bronchial irritation by several days and the phrase is current "th
cold has gone on to the chest."

In this class of cases the extension of the inflammation is due to the action
of micro-organisms which have found a suitable culture medium in the altered
secretions of the nares or throat. Streptococci are the germs commonly found,
and on their virulence depends the intensity of the process. It is generally
found, I think, that the nasal passages are not in a healthy condition even
when no acute coryza is present. There is a chronic atrophic or hypertrophic
rhinitis and this in itself predisposes to acute inflammation and hence to
bronchitis. Frequent repetition of the acute is apt to lead in time to the
chronic form.

Exposure.—Although it is by no means necessary that an individual who
develops an acute bronchitis shall have exposed himself to a chilling of the
body, or to a wetting, still it is a very common experience that a com
the subsequent bronchitis follow an exposure to sudden
or temperature.

Persons are often surprised by the appearance of a cold in the head or on the chest who have not been outside the house for days, and then they wonder how they contracted their cold. For my part I believe that in many instances the local inflammation is the result of some local irritation, as dust, or of some constitutional influence which favors the action of germs constantly present in the nares or throat, or that the process is a true infection and that an antecedent exposure is not at all necessary.

In other instances on the contrary the bronchitis, whether purely acute or an exacerbation of a chronic bronchial catarrh, does actually follow an exposure of some kind. Such a predisposing condition may be the sudden cooling of the surface of the body to which one is liable on leaving an overheated building on a raw winter day or one of the extreme and sudden falls in temperature so characteristic of some localities in the Spring or Fall. Some individuals are almost certain to get up a coryza and subsequent bronchitis from a wetting of the feet, a draught of cold air on the back of the neck or uncovered head, especially if at the time they are in a hot, ill-ventilated apartment or public conveyance.

It is not quite easy to explain the injurious effect of exposures of the kind just mentioned, unless we invoke the aid of bacteriological teachings. If an individual has been breathing an overheated, ill-ventilated and hence germ-laden atmosphere, he has been subjecting his mucous membranes to conditions especially favorable to the development of inflammation. In consequence of the passage of hot, dry air over his respiratory mucous membranes there has taken place a dilatation of the blood-vessels of the part while at the same time an abstraction of moisture has occurred. The vessels are, to a degree at least, in the same state of paresis as are those of animals that have been subjected to heat in an air-tight box. When now the animals are suddenly immersed in cold water an abstraction of body-heat takes place. To prevent such a sudden and injurious withdrawal of body-heat the superficial vessels become contracted and the blood is thrown inward.

In the case of human beings who are suddenly exposed to local or general chilling, a fluxionary hyperæmia takes place in the part of the body which is of least resistance. What is more natural than that such a congestion should occur in the nasal or tracheo-bronchial mucous membrane which has been suffering from irritation and dilatation of its superficial vessels?

In fact this is exactly what does take place. Moreover, the nutrition of the mucous membrane has been impaired previously or now becomes impaired, and the epithelium cannot resist the action of the germs that were previously present or have been lodged thereon out of the impure air just inspired. The conditions predisposing to irritation and inflammation are all furnished. The individual very likely has some local defect, either a chronic rhinitis or a chronic congestion of the bronchi or a tendency to pulmonary inflammation in consequence of some other of the predisposing causes subsequently to be mentioned. Hence, as soon as he is subjected to exposure he begins to snuffle or sneeze or to experience a feeling of tightness of the chest, in short some sensation which tells him that he has taken cold.

Defective Ventilation.—This is a condition which according to
of the bacterial origin of bronchitis must be regarded as a highly
predisposing factor. Such an impure atmosphere is laden with in
organisms, some of which may be pathogenic, and hence the persor
remaining for several hours in such an atmosphere is subjected t
sibility of their lodgment and growth on his mucous membranes.
that such is the explanation of why babies and old persons some
tract colds and coughs who have not been outside the house all wir
tainly pure air is a preventive against many diseases and why n
bronchitis?

Chemical and Mechanical Irritants.—Direct local irritation i
well-recognized factor in the production of acute as well as of chr
chitis. The inhalation of such irritants as fumes of Bromine, Chlor
Acid and even the smoke or gas from a locomotive or furnace, ma
violent inflammation so speedily as to class this inhalation amon
causes. It is more scientific to regard it as a predisposing cause sinc
but to prepare the soil for the action of bacteria either by settin
hyperæmia or by affecting the nutrition of the protecting epitheliun

Fog, especially a London fog, is a capital example of the pr
influence of atmospheric impurities in causing acute bronchitis. I
repeatedly observed that a week of dense fog in that city would be
by an enormous rise in the death-rate and that this increase in tl
of deaths was due almost entirely to acute bronchitis.

A London fog does not consist merely of vapor, but the tiny
moisture are coated over with soot which renders them exceedingl
to the effect of heat within doors and extremely irritating to the ai
Moreover, such a foggy atmosphere must furnish an excellent m
the conveyance of organisms to the bronchial mucosa, and when
these would be likely to profit by the nutritional disturbance or oth
induced by the irritating properties of the fog, and excite an acu
mation.

Whereas a London fog may be a powerful factor in the productio
bronchitis *de novo* or as an exacerbation of a chronic affair, it is w
that the daily respiration of an atmosphere laden with smoke or d
poses to the development of chronic bronchitis independent of acu
It does so probably by keeping up a constant irritation of the epitheli
in time leads to its defective nutrition, or by inducing a hyperse
mucus in which the germs may grow and multiply. It is for the sa
that a dusty atmosphere is also injurious as will be shown in c
occupation.

In this connection may also be mentioned the injurious influence
days in large cities in setting up acute catarrhal inflammation of tl
derian or tracheo-bronchial mucosa. In most large centers of popu
streets are never so clean as to prevent the impregnation of the ai
tensely irritating dust on windy days. Not only are the dust partic
composed of mineral, as stone and iron, but they carry with them in
bacteria of all kinds. Consequently, as is well recognized, a spell of

weather is always followed by an increase of nasal and bronchial catarrhs. It is on this account in part that the inhabitants of smoky and dirty cities are more likely to suffer from chronic bronchitis than are the residents of rural districts, who are not predisposed by such atmospheric impurities.

Tobacco smoke is another of the irritants that may conduce to the production of chronic bronchitis. It may seem strange to include tobacco smoke in the category of predisposing causes, yet, that it does irritate the mucous lining of the trachea and bronchi is shown by the cough so promptly excited when the smoke is inhaled. Nevertheless, this form of smoke is not injurious to the lower respiratory tract under ordinary circumstances. Its harmfulness comes when it is habitually inhaled, as it is by many cigarette smokers. They may establish a certain degree of immunity so that they do not cough upon inhaling the smoke, but the epithelial covering of the mucosa is irritated and more or less congested. It is this state of the mucous membrane which in time may lead to chronic inflammation.

Another condition under which tobacco smoke becomes harmful to the air-passages is that to which Liebermeister directs attention. He dwells in particular on the irritation to which the air-tubes are exposed when persons are in the habit of spending evening after evening in the smoke-filled atmosphere of taverns and barrooms. In these and kindred places of resort the air becomes literally saturated with smoke from strong pipes and cigars, so that with every breath the *habitué* draws the irritant into his lungs. In addition he generally consumes more or less liquor, and this, as will be shown, directly intensifies the injurious effect of the tobacco smoke. Moreover the air of these places of resort is impregnated with organisms which are carried with the smoke into the air-passages.

Alcohol.—As is well known, chronic bronchitis is frequent among old and hard drinkers. There are doubtless many factors at work here, but no insignificant place must be given to alcohol *per se.* This substance is eliminated in part by the lungs, and hence the mucous lining of the bronchi is habitually subjected to its irritant action. It is for this reason that tobacco smoke and alcohol together predispose the saloon-lounger to chronic bronchitis, and that very few such persons escape the disease, which, in them is particularly obstinate.

Drugs.—There are certain medicaments which are capable of exciting acute bronchitis when taken in large continuous doses. These are the salts of Iodine and Bromine. They are eliminated by the mucous membranes and hence are capable of irritating the tracheo-bronchial mucosa as well as that of the nares. All persons are not equally susceptible to the action of these chemical irritants. Some are especially sensitive to even small doses of them, and it is conceivable that if such drugs were continued for a long time they might at length conduce to a chronic inflammation of the bronchial mucosa.

Previous Diseases.—Most of these exert a predisposing influence through mechanical congestion and hence bronchitis is frequent among sufferers from chronic heart disease. Mitral patients are far more prone to it than are persons with aortic valve lesions, for the reason that mitral disease, especially stenosis, is attended with more or less stasis within the pulmonary and bronchial veins. Such cardiopaths frequently have cough with sligh expectoration even when they do not present signs of actual bronc

I have frequently seen mitral sufferers develop attacks of acute but, unless they were subjected to other factors, they did not present of chronic bronchial inflammation. On the other hand I have very observed a chronic bronchial catarrh in persons who had arteri chronic myocarditis or both of these conditions, either with or witho nephritis. In these diseases there is also more or less pulmonary and stasis, the same as in mitral disease. Yet as the former are often with chronic bronchitis, while mitral lesions are not, there must be s tional factor also at work.

These additional factors must be the action of bacteria. Else v see so much less bronchitis among persons who, having cardio-v renal disease, are yet well housed and environed, while it is extremel among hospital patients suffering from identical chronic disorders. uals belonging to the better class are better nourished and are a more orderly lives, and hence can more successfully resist the action isms. Nevertheless, when they contract an influenza or an acute str bronchitis there is a marked tendency for the infection to becom doubtless on account of their original cardiac, vascular or renal di

Gout and Syphilis likewise are diseases which are said especial dispose to both acute and chronic bronchitis. The former is atte circulatory disorders that in themselves predispose to the affection, sufferers are peculiarly liable to acute congestions if not to actual tions of the various mucous membranes. Therefore something r mere stasis is at work within the bronchial vessels. It seems to me q that the blood of gouty persons contains some chemical irritant urate of soda) that is eliminated by way of the various mucosæ renders the air-tubes especially liable to inflammation in the p bacteria.

Syphilis certainly manifests a predilection for the mucous su hence there is in this infection an undoubted tendency to the pro chronic pulmonary and bronchial disorders. I have never observed w be called a syphilitic bronchitis but I have often noted that perso hospital inmates, suffering from chronic bronchial catarrh give a a chancre years previously. Lang is said by Fraenkel to have obse bronchial catarrh coincidently with the primary exanthem and wh peared with the eruption. Such instances are exceptional, howev bronchitis is usually of the chronic sort.

Pulmonary Diseases.—There are certain diseases of the lungs so uniformly attended with bronchitis, sometimes acute but usuall that they must be reckoned among the predisposing conditions. T these is vesicular emphysema and closely associated therewith is asthma. In both of these affections chronic bronchial catarrh is a tain to develop in the course of time. In the former it is probabl gestion together with the frequent occurrence of acute bronchial infl that especially predisposes to chronic catarrh, while in asthma the ca not become manifest until after emphysema has been produced. It is likely therefore that in this affection also the chronic bronchitis is referable to stasis and impaired nutrition or to some alteration in the quantity or quality of the mucous secretions or to both.

Cirrhosis of the lung, with or without bronchiectasis, also predisposes to acute and chronic bronchitis. The cause probably lies in the presence and action of germs which for some reason find these diseases particularly favorable to their growth. More will be found on this subject in the respective chapters.

Pulmonary Tuberculosis.—This disease is always accompanied by more or less bronchitis which may be bilateral and general, or what is far more common circumscribed to the immediate proximity of the tuberculous area. In this affection the bronchitis is unquestionably due to the action of the specific bacillus or to associated organisms, as streptococci, which as they pass over the bronchial mucosa in the act of expectoration set up inflammation. The bronchitis may be said to be an example of disease by extension from below upward. In cases of fibroid phthisis it is the chronic bronchial catarrh that very largely maintains the cough and expectoration.

Occupation.—The influence of occupation is seen in the frequency with which persons following certain employments suffer from chronic bronchitis. The injurious effect of occupation is attributable mainly to the inhalation of dust incident to certain vocations. Hirt, whose work is an authority on this subject, has shown that the inhalation of vegetable dust is the most liable to occasion chronic bronchitis, while animal dust is the least so. Metallic and mineral dusts come next in the order named.

Whereas the inhalation of vegetable dust is liable to produce bronchitis, the more irritating metallic and mineral dust is likely to lead to a form of phthisis that was formerly supposed to be different from tuberculous consumption. (See Pneumoconiosis.) It is now known that when consumption develops among workers in dust, it is owing to the presence of tubercle bacilli and that the pneumoconiosis only serves as a predisposing factor. Therefore, the inhalation of vegetable dust predisposes to the production of chronic bronchitis through the irritation produced by the dust particles and the growth of organisms carried in along with the dust. Accordingly, millers and grain-shovelers are especially liable to bronchitis while next in order come weavers and workers in cotton factories.

The depth to which minute dust particles may penetrate the lungs is shown by their deposit in the walls of the alveoli and infundibula and by their discovery in the lymph nodes at the root of the lungs to which they have been carried by the lymph stream from the alveolar passages. The coarser particles lodge in the larynx and trachea and, causing irritation, are expelled by the act of coughing they excite. The major portion of finer fragments is deposited upon the mucosa of the primary bronchi and adhering there predisposes to the development of chronic inflammation.

Even in the case of dusty occupations I believe the actual determining factors in the ætiology of bronchitis are the bacteria carried along with the dust. The tardier development and less [illegible] character of the inflammation they excite may be explicable on the supposition that although the mately impairs the nutrition of the bronchial epithelium, it yet f protects this latter from the injurious action of the germs.

Cigarette smoking is in a similar manner, I believe, capable of pr to the production of chronic bronchitis. The inveterate cigarette fie his tobacco smoke and this as everybody knows is a powerful irrita cous membranes. In addition it undermines the general health, i. tissue-resistance to invading organisms, and thus exerts a doubly influence.

Age.—Acute bronchitis is met with at all ages but by no mea quently in the middle years of life as at either extreme. Children cially liable thereto for a number of reasons. The occurrence of spe chitides in children depends upon their greater susceptibility to m pertussis, but even as regards nonspecific bronchitis they manifest vulnerability which is in large measure due to their diminished res all kinds of predisposing influences. In particular Fraenkel calls to the shortness and narrowness of the air-passages which are thus susceptible to injury from cold air when inhaled.

Old persons are likewise prone to attacks of acute bronchitis. there is also a feeble resisting power; but the chief predisposing in their arteriosclerosis and their weak hearts. As previously po these conditions lead to congestion within the lesser circulation and ency to stasis is a decided predisposing element.

Children may develop chronic bronchial catarrh with its c emphysema of the lungs and dilatation of the right heart, especia age of six or seven (Fraenkel). Nevertheless the liability t bronchitis increases with advancing years and attains its max old age. The reasons therefore are to be sought in the changes vessels, kidneys and even lungs (emphysema and atrophy) th senility.

Season and Climate.—These undoubtedly play an important r production of both acute and chronic bronchitis. These affections cially prevalent in those seasons and in those climates in which su violent changes of temperature abound. Hence they are most frequ Fall, Winter and Spring months and in the temperate zone.

At these seasons and in countries occupying the temperate reg are not only sudden and extreme transitions from warm to cold d with high winds, but there is great relative and absolute humidity, cold days the wind is raw and penetrating.

Such weather is not only directly trying to the nasal and bronchi membranes, as shown by the prevalence of catarrh, but persons p from a warm perhaps ill-ventilated building are very likely to feel chill. This leads to a sudden congestion of some vulnerable mu brane, as was pointed out under exposure, and thus bronchial cat acute and chronic is occasioned. In a word the evil effects of ex most often experienced in inclement seasons and in changeable Moreover at such times and in such climes there are added the effects of prolonged sojourn in overheated and imperfectly ventilated buildings, as was previously stated.

Morbid Anatomy.—Any portion of the bronchial tree may become inflamed, but what is known as acute simple bronchitis generally affects the trachea and the larger and medium-sized tubes of both lungs. It is therefore a tracheobronchitis and is bilateral. The inflammation is rarely severe enough to invade the deeper structures, but is limited to the mucosa. The initial changes are hyperæmic redness and swelling. The hue imparted by the congestion is rarely deep, but may be of a purplish shade. This color is apt to fade out after death, leaving the membrane pale.

The hyperæmia of the vessels in the inner fibrous layer tends to more or less occlusion of the ducts of the mucous glands. Hence in the early stage of inflammation the surface of the mucous membrane is dry and the slight quantity of mucus present is thick, viscid, opaque and mixed with epithelial cells. As inflammation grows less active mucus is secreted in large amount and assumes a more fluid and purulent character from the admixture of serum, leucocytes and a scanty number of red blood cells.

The microscope reveals thickening of the epithelial layer in consequence of active proliferation of the cells underlying the columnar ciliated epithelium which latter is more or less degenerated and in a process of desquamation. In the more active forms of inflammation there is also apparent a cellular infiltration of the inner fibrous and muscular layers; but although some degree of such cellular infiltration attends every case of acute bronchitis, it does not lead to permanent changes in the walls of the tube.

When acute inflammation involves also the finer bronchi these are apt to become blocked by the secretions with resulting collapse of the lobules to which the tubes lead.

The secondary changes discovered in other viscera are not marked and consist of such alteration as results from congestion. In cases of severe and widespread bronchitis the right heart becomes engorged and dilatation of the ventricle and tricuspid ring leads to relative incompetence of the valve.

In *chronic bronchitis* the changes discovered are of a far more serious nature and include the walls as well as the lining membrane of the tubes. In some cases one is struck by the paucity of pathological lesions as compared with the clinical history, while in others the changes are pronounced and widespread. The mucous membrane may be thinned and pale or thickened and congested. If the case has been one of dry catarrh the tubes may contain but a scanty amount of tenacious mucus and the glands may be atrophied. Usually however there is a considerable amount of mucus mixed with pus which having undergone decomposition from the action of putrefactive organisms gives forth a very offensive odor (fœtid bronchitis).

The mucous glands are congested. The lining epithelium is degenerated and in the process of desquamation. The internal fibrous layer is infiltrated with cells and the external layer of connective tissue (adventitia) is thickened (hyperplastic). The muscular layer is also thickened and when such hypertrophy is marked, the inner surface of the tube is thrown into longitudinal and transverse ridges.

In places the lumen of a bronchus may be narrowed in cons
hyperplasia of the fibrous layer, while just peripheral thereto a
dilatation may exist (bronchiectasis). The coats of the bronchi
are also apt to be thickened (Delafield and Prudden).

The changes of chronic bronchial inflammation are almost invar
ciated with vesicular emphysema for the reason that whichever p
cedes it is quite certain in time to lead to the production of the oth
pulmonary lesions likely to be found associated with bronchitis are
pneumonia, chronic tuberculosis and old pleuritic adhesions.

Cardio-vascular and renal disease is also common. There may
disease with brown atrophy, or there may be chronic myocarditis wit
lesions. In either event the right heart is apt to be dilated, its w
trophied and the tricuspid ostium stretched. It is not rare to dis
siderable, even extreme, sclerosis of the pulmonary artery, which
may be either a sequel to or a cause of the bronchitis.

The aortic system is very apt to be sclerotic, the liver in a state
induration, other abdominal viscera congested, with the kidneys
changes of chronic nephritis.

CHAPTER II

ACUTE BRONCHITIS

Symptoms.—The bronchitis observed in the course of specific infections forms a part of the clinical picture of those diseases and requires no especial description. The feature that generally attracts particular notice to the bronchial complication is cough. This may be mild and occasion no great annoyance, or if the inflammation is intense and widespread it may be both frequent and severe. Such is very apt to be the case in the bronchitis of *grippe* and in that of measles and pertussis in young children. In the last-named affection it is so characteristic as to have given the popular designation to the disease.

There is usually expectoration of a variable amount of mucus which is more or less viscid and contains the organisms responsible for the bronchitis. The sputum also shows epithelial and alveolar cells, sometimes a few red blood cells and leucocytes. If the latter abound in sufficient numbers they impart a yellowish hue to the expectoration, when it is spoken of as muco-purulent or purulent.

If there is any increase or modification of temperature due to the bronchial catarrh it is generally disguised by that of the primary affection. The pulse is usually unaffected by the bronchitis, unless possibly through the violence and frequency of the cough, and the respirations are likewise but slightly influenced. In young children and in cases in which the bronchitis has widely involved the smaller tubes, the breathing is likely to be accelerated. If the condition be what is sometimes spoken of as capillary bronchitis (see Bronchopneumonia) the dyspnœa may become so extreme as to merit the old term Suffocative catarrh. There is then more or less cyanosis, restlessness, anxiety of the countenance and feebleness.

In mild cases the bronchitis may occasion no other symptoms than cough and its existence is only determined by examination of the chest. The resonance of the lungs is generally retained, any impairment that may be detected at the posterior base being occasioned by some associated and dependent condition, as collapse or broncho-pneumonia. On auscultation there may or may not be whistling and fine bubbling râles either general or circumscribed which partially or wholly obscure the breath-sounds. When these latter can be recognized, they are roughened or possibly diminished in intensity but are not bronchial. In typhoid patients it is the recognition of these objective signs rather than any general symptoms that enables one to determine the existence of the bronchitis.

In the young, the aged and the feeble, such a specific bronchitis may prove

a very dangerous condition either *per se* or by the atelectasis
pneumonia to which it leads.

An active healthy lad of nine and a half years began on Nov
to show signs of acute coryza for which his mother, supposing
ordinary cold in the head, gave him calomel. The next day his
on in full force, but he did not appear ill. On the third day hi
hoarse and he began to have a dry barking cough. At noon he con
slight chilliness and ate almost no luncheon. In the afternoon he
to take a short walk but returned saying he was cold. His temp
taken and found to be 103° F. The lad was then put to bed and
was summoned.

Examination revealed nothing more than coryza, slight redn
pharynx and pyrexia. On the fourth day his condition remaine
statu quo, but on the morning of the fifth day it was evident th
had an acute bronchitis which was rather more marked on the left
being a few faint sibilant râles at the extreme base behind. It
tolerably certain that something more was present than a mere e
the coryza. Accordingly, cultures were made from the secretions of
and from a little sputum that was raised with some difficulty, a
in the growth of typical influenza bacilli.

A few days later the continuance of the pyrexia and the appear
tongue created the suspicion of still another infection, namely ty
which abounded in Chicago at the time. Accordingly, blood was
examined for the Widal reaction. Clumping was not typical
sufficiently pronounced to warrant the conclusion that the la
suffering from typhoid. The course of the fever was mild; but
bronchitis increased and eventually yielded signs of its being
eral, although broncho-pneumonia did not develop and a goo
was made.

The subsequent history of this case is so instructive as to justi
narration. As stated, the lad convalesced from his specific bro
apparently atypical typhoid. In the fore part of January he cam
afternoon complaining of intense pain in the right iliac fossa. H
ture was 101° F., the abdomen at that part was tender to pressure
rigidity and he had a pronounced leucocytosis. The attack was
of acute appendicitis. A successful operation was performed ne
by Dr. Bayard Holmes.

The appendix was bound by old adhesions, held at its tip s
cheesy pus containing colon bacilli and furnished signs of acute inf
Careful inquiry was now made of the lad and he then recalled
Saturday afternoon when he had experienced a chill and had bee
have fever, both of which were attributed at the time to bronchi
had pain in the abdomen. In the light of these facts it was now b
his atypical typhoid was in reality an acute appendicitis. It is the
esting since it has been claimed that appendicitis not infrequentl
grippe infection. Influenza bacillus was carefully sought for in the
appendix but not found.

Symptoms of Acute Nonspecific Bronchitis.—These may vary greatly in severity from so trivial a disturbance as scarcely to attract notice, to one of such intensity that the patient is confined in bed, and, if he recovers at all, does so with difficulty. In the great majority of cases the trachea as well as the main bronchi is affected and the process is an extension downward of a coryza or sore throat. In the minority of cases the bronchitis starts as a primary affection and follows more or less quickly an exposure of some sort, as a chill, a draught on the back of the neck, or a wetting of the feet.

Whatever may be the mode of onset, the earliest symptoms are a growing sense of *tightness in the chest* and a frequent desire to *cough*. One of my patients first becomes conscious of having taken cold by what she describes as an itching and feeling of tightness which begins in the lower part of the neck and as it grows worse extends downward until it reaches all the way down the sternum to the pit of the stomach. Her sensations may be said to describe very well the initial manifestation of an acute tracheobronchitis.

Cough is another early symptom which may attend the sense of constriction from the start or may set in somewhat later. At first the cough is short and dry, unproductive, as it is called, and usually produces a feeling of pain beneath the sternum which is of a cutting or burning character and leaves the chest feeling tighter than before. Instead therefore of relieving the patient cough appears only to aggravate his sense of substernal oppression and to intensify his desire to cough afresh, so that he is impelled to make strenuous though usually ineffectual effort to keep down the cough.

It is very common in this initial stage for strong, healthy adults to experience nothing more than a slight *malaise* and to display no rise of temperature or other evidence of constitutional disturbance. Debilitated subjects on the contrary, the aged and young children, are very likely to appear more or less ill and to complain of "aching in the bones" or pain in the back and head.

If examined at this time, the pulse is found accelerated and the temperature raised a degree or two depending upon the intensity of the inflammation and the reaction of the patient. Young children in whom febrile reaction is always more marked than in adults, not infrequently exhibit a temperature of 102.5° or 103° F. and a rapid bounding pulse with hurried respirations. It is the pyrexia probably, therefore, that accounts for the pain and aching in various parts of the body. In cases displaying such symptoms of constitutional disturbance there is also some degree of anorexia, constipation and scantiness of urine, with perhaps thirst.

After these symptoms have persisted a variable number of hours, depending on the activity of the process and the institution or not of treatment, the feeling of substernal weight and soreness abates a little and the cough becomes looser and less frequent. The patient now begins to raise a scanty expectoration which consists of viscid, glairy mucus, rich in mucin and containing a few desquamated epithelia and micro-organisms. In some instances, when cough is especially violent and prolonged and the inflammatory congestion more than ordinarily intense, the sputum may contain a little frothy serum or even be streaked with blood. It is then likely to have a distinctly salty taste.

With the establishment of expectoration the cough generally lessens in frequency if not in severity, yet this depends largely upon the por respiratory tract which bears the brunt of the inflammation. Ko ments demonstrated that cough is most prompt and severe when i applied to the interarytænoid fossa and the bifurcation of the trach the mucous membrane of the windpipe and bronchi is less sensitiv still capable of originating reflex cough. Consequently if sev spasms still persist after the intensity of the tracheobronchitis they may generally be taken to indicate that the brunt of the borne by the mucosa lining the trachea at its division or that t membrane is still irritated by the presence of tenacious mucus. It of course that the interarytænoid fossa or some of the folds of mu brane connected with the epiglottis have been involved by the in process, or congested and irritated in consequence of frequent acts o Therefore the physician should not fail to examine these parts wh symptom remains unusually prominent.

With further abatement of inflammatory hyperæmia and loose cough the sputum changes in character and appearance. It is still mucoid but assumes a more or less yellowish color from the ad cellular elements, chiefly pus cells but also others that have unde degeneration. It is now spoken of as muco-purulent or purulent to the proportion of mucus or pus it contains. Micro-organisms are dant and usually consist of streptococci or staphylococci or both.

The expectoration in this stage varies in amount, but is as a rule especially in the fore part of the day. Cough is apt to tighten up approaches and may be difficult before breakfast; but after the breakfasted he generally succeeds in clearing out his tubes after wh be free from expectoration for several hours.

Coincidently with the change for the better in these two symp is progressive diminution of substernal pain upon the act of cou temperature, if elevated, returns gradually to normal, the pati strength and appetite and after one or two weeks from the onset no further discomfort than a slight morning cough. The air-passa to remain unduly sensitive to sudden transitions of temperature, from a warm house into the cold outside air, but this also wears av and recovery is complete.

One occasionally encounters cases of acute bronchitis so decided ter as to create a suspicion of something more than a mere bronchi the course and duration of the affection bear out the accuracy of th Such an instance was seen recently in my service at Cook Count The case illustrates moreover the ætiology of some of the instan persons who lead an irregular mode of life.

An Irish laborer, aged forty-two, was admitted to Ward 24 S 1905, on account of cough and dyspnœa. He was a powerfully bui admitted having been drinking heavily for two weeks. One week be sion he had had an acute coryza and this had been followed by a c year ago he had had an attack similar to the present one which a lowed a prolonged drinking bout.

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lowed a prolonged drinking bout.

On examination it was found that he had a temperature of 101.8° F. but his pulse was 96 and his respirations were 24; the pulse-respiration rate therefore was normal. The man was coughing very frequently and complained of pain in the right chest in front on cough; he could not raise sputum for examination. The percussion note was everywhere resonant and yet the right upper lobe behind seemed a trifle higher pitched than did the opposite lung. Auscultation disclosed throughout both lungs a multitude of whistling and gurgling râles. The heart and urine were negative.

The case seemed plainly one of acute bronchitis and the man was given a dose of magnesia sulphate and an expectorant mixture containing chloride of ammonium.

The next morning the temperature fell to 98.8° F. and thereafter remained at about that figure, going on one or two occasions to 99° F. The râles soon changed, those in the left lung practically disappearing in a day, while at the right some moist râles were recorded as present on the second day after his admission. September 6th he felt so well that he sought and obtained his discharge.

This case illustrates very well the inception, symptoms and course of acute bronchitis in a robust man. Still, had the man's habits been better, it is likely that the affection would not have assumed so marked a character as it did. Especially there would not have been so many and so pronounced râles. Finally it is precisely this sort of a man who in time develops a chronic bronchitis out of the frequent repetitions of acute attacks; particularly when the disposition to bronchial catarrh is kept up by whisky and tobacco added to the exposure incident to his work as a laborer.

The symptoms and gravity of acute bronchitis in an individual suffering from cardiac disease are well exhibited in the following case in which I was acting as consultant. A portly gentleman of fifty-two who resided at Riverside, a suburb of Chicago, was confined to his room with cardiac incompetence, probably from chronic myocarditis. The clinical picture was that of anasarca, a feeble rapid pulse, general cardiac dilatation with a mitral systolic murmur, and the signs of relative tricuspid insufficiency, i. e., the characteristic murmur and positive venous pulse in the right external jugular.

He was seen in consultation with Dr. Sterl for the second time on Thursday morning, December 18th, and was found to have improved somewhat on cardiac tonics, cathartics, rest in bed and resistance exercises. Nothing was said at the time about his having taken cold, as he supposed, although as was afterwards learned, he was having "a little catarrh of the throat." Saturday afternoon, two days later, I was notified by telephone that the patient had a bronchitis and was so much worse that a consultation was desired at once.

At 5 o'clock on December 20th I found his condition as follows: The man was in bed in the dorsal decubitus, breathing rather heavily 26 to the minute, with loud whistling audible at a short distance away, pulse 112 and feeble, temperature in the mouth 99.9° F. The heart was apparently the same as two days before. The lungs were everywhere resonant; but in front, especially over the main bronchi, were loud whistling and sonorous râles which in great measure obscured the breath-sounds. So far as could be determined,

however, there was no bronchial breathing. At the bases, behind,
tory murmur was exaggerated and accompanied, but not obscure

The condition seemed to be a widespread acute bronchitis w
monia, although the possibility of this latter developing was kept

As the patient had not slept for two nights he was given a
injection of morphine, and in addition digitalis and strophanthu
what increased, a sixtieth of strychnine was ordered subcutaneousl
in twenty-four hours, and a cough mixture was prescribed conta
teenth of tartar emetic with five grains of ammonium chloride
He passed a comfortable night and during Sunday expectorated
Sunday night without morphine was spent in fitful sleep and M
ing he felt weak and depressed in spirits.

At my visit on this morning his condition appeared to me abo
been Saturday afternoon. The heart was holding up fairly well
perature by mouth was normal. The lungs however were full
and sibilant râles even to the extreme base in front and behind.
careful search failed to disclose dullness or bronchial breathing.

Tuesday morning as a result of prompt treatment his conditi
satisfactory. Temperature was 99° F., pulse was 100 and of bet
respirations were less wheezy and the duskiness had disappeared.
revealed no dullness and the râles were less noisy and numerous.
had excreted 48 ounces of urine besides having had three liquid

Improvement continued during Tuesday, expectoration was
Wednesday morning but a single inspiratory rhonchus was audi
out the chest. The heart had gained in strength, and pulsation
external jugular was less pronounced. It was evident that the
bronchitis was over and that recovery from this complication was

I was curious to ascertain the cause of this attack and upon mi
learned the following. A baby belonging to the wet nurse had an
fested by an eruption on the head and arms, with fever and bronchi
or two subsequently this gentleman's fifteen-months-old grand
oped a coryza and loose catarrhal cough. This child visited his
daily and often sat in his lap. Two days prior to the occurrence
chitis this gentleman contrary to orders went downstairs and sa
where he was exposed to a draught in consequence of which he be
chilled. The next morning he noticed irritation of his throat and
ing evening, Friday, he began to wheeze somewhat. By Saturd
chitis was unmistakable and that afternoon I was summoned to
as narrated. It is my opinion that he became infected from his
and that it only required exposure in his weakened state to act a
mining cause of his bronchitis. Its fortunate termination was d
the promptness with which treatment was instituted.

The gravity of bronchitis in elderly men was well illustrated
of Col. M— whom I saw in consultation with Dr. Hollowbush of
at the patient's home in October, 1902. The colonel was a man
physique and of great activity who, aside from being wounded
War, had never known illness. Consequently, when in March, 19

oped a cough, he thought the cold was not of much importance and neglected treatment. Whether this cough developed as an acute tracheo-bronchitis is not quite clear, but it came on shortly after the colonel had perceived such an unwonted shortness of breath on walking uphill one blustery March day as to compel him to sit down to recover his breath. He may therefore have been heated and suffered a too rapid cooling off, to which, being a vigorous man he paid no heed.

At all events he went on without treatment and without throwing off his cold on the chest until August, 1902, when he was led by his asthmatic breathing to consult Dr. Hollowbush who discovered signs of generalized bronchitis. The chest was full of dry râles, cough was violent and spasmodic, expectoration consisted of stringy mucus; temperature was normal and the heart appeared healthy.

In October the condition had taken a turn for the worse and the doctor sought counsel. I found that for five nights the patient had not slept without an opiate, and that his great restlessness, difficulty of breathing, paroxysmal, violent cough, abundant sometimes bloody sputum, feeble arrhythmic pulse had presented a clinical picture well calculated to create apprehension and had rendered frequent stimulation with whisky necessary.

My examination revealed great respiratory and cardiac embarrassment, the latter being shown by relative tricuspid insufficiency. There was a mouth temperature of 101.8° F., and in the lungs there were areas of dullness due to atelectasis and pneumonia, with innumerable sibilant and subcrepitant râles which nearly obscured the breath-sounds. In a few areas however bronchial respiration and true crepitant râles made it evident that pneumonia had in the last few days been added to the preëxisting bronchitis and atelectasis.

The patient felt so vigorous in a muscular way that notwithstanding his dyspnœa and cough he could with difficulty be impressed with the necessity of remaining quiet in his apartment. This was insisted upon as a *sine qua non* of recovery and thanks to the mildness of the pneumonic infection, the natural vigor of his constitution and good nursing, after two weeks he was reported as convalescing satisfactorily.

In this instance my uneasiness was enhanced by the discovery of stiff peripheral arteries, an intensely booming aortic second tone and an increase of deep-seated cardiac dullness that pointed to dilatation as well as hypertrophy, all changes, which betokened chronic myocarditis. Although this gentleman was still vigorous at sixty-two, his citadel, the heart, had been undermined and no one could say how seriously. The tricuspid regurgitation was a measure of the strain to which the right ventricle was subjected and was thought to be an omen of grave import. The bearing of this relative incompetence in cases of bronchitis will be discussed in the chapter on Chronic Bronchitis.

The foregoing case illustrates in several ways the gravity of bronchitis in elderly men and teaches some instructive lessons. In the first place, that which in the beginning is but a simple bronchial catarrh may very readily ass into a chronic condition. Men of the type of this one generally have stiff rte [illegible] chronic myocarditis although the heart may be potentially equal to its every-day work. There is therefore more or less congestion of the pulmonary and bronchial vessels which predisposes these individuals to bronchitis and when this has once been established renders it intractable to ordinary methods of treatment. Moreover, such previously strong men are apt to think they can easily " throw off " their colds and hence do not seek medical aid until the condition has become settled and obstinate. When at length the physician is consulted he is too prone to look upon the ailment as trifling and not to recognize the cardio-vascular changes which influence the prognosis. An expectorant is prescribed and the patient is permitted to pursue his usual vocation, with the result that the bronchitis does not yield as was expected.

The second element of danger lies in the possibility of pneumonia. In one set of cases the chronic bronchitis predisposes to the development of pneumonia as in the case narrated, while in another and far more dangerous group the bronchitis which sets in abruptly and severely is associated with and is in reality an attendant of a pneumonia which, because of the bronchitis, is not discovered. In the light of this possibility, the physician should regard acute bronchitis in elderly men as sufficiently serious to warrant careful attention, both on his part and on that of the patient.

A third element of danger to elderly men consists in the state of the heart-muscle. Chronic myocarditis develops so insidiously as to be often unsuspected until cardiac incompetence asserts itself, during some illness like bronchitis, which in a younger individual would prove a comparatively trifling matter. It is the effect upon the heart's potentiality which lends gravity to the existence of myocardial decay, and the degree of reserve power still possessed by the organ cannot be foreseen. For this reason it is safer to underestimate than to overestimate it, and in a case of the kind here reported the heart should always be sustained. I do not mean alone that digitalis and strychnine are to be administered, but that the patient is to be informed of the possible dangers and is not to be allowed to add to his cardiac strain by keeping about in the performance of business or for the sake of exercise. Other things being equal recovery from the bronchitis is more rapid and certain if the patient remains quiet at home or even in his apartment when the pulmonary complaint is severe.

Finally, if in a given case overstrain of the right ventricle leads to relative tricuspid insufficiency, the regurgitation should be regarded not as a sign of danger *per se* but as a criterion of the strain felt by the heart. The tricuspid leak acts as a safety valve, but only within certain limits. If the cardiac overstrain persists too long or passes beyond a certain degree, the myocardium will be forced to succumb in spite of the aid afforded by the giving way of the tricuspid valve. In not a few cases the safety-valve action of the tricuspid leak furnishes the overburdened heart a temporary relief and affords the physician a little longer time in which to assist the heart by his remedies. It cannot fail to make the medical attendant anxious, but it may at the same time give him hope of winning in his struggle against the adverse forces of the disease.

Duration and Termination.—An ordinary case of acute bronchitis in a healthy adult generally lasts two or three weeks. It does not terminate abruptly but gradually grows less pronounced in the symptoms that indicate inflammation. The persisting morning cough and expectoration are but the remnants of the trouble, so to speak, and these are likely to disappear in a fortnight or thereabouts.

If recurrences take place as is not infrequently the case in the late Fall before the original bronchitis has entirely subsided, the duration may be greatly prolonged and more or less bronchial sensitiveness and irritation may continue through the Winter. A robust individual may then succeed in throwing off the disease for good, but in feeble persons or such as have some predisposing affection of other organs there is strong probability of the catarrh becoming chronic.

In children and old people an acute bronchitis is not a simple matter. Even if not due to influenza or some other specific infection it may readily prove more serious than their limited resistance can endure. Nowadays I rarely see what may be called a case of simple acute bronchitis in children or in persons past middle age. The disease is generally one that by its persistence and other characters as well as by the sputum examination is seen to be plainly a manifestation of specific infection of the air-tubes. Consequently it cannot be considered as a cold on the chest but is symptomatic of an influenza or pneumococcus infection and shows a marked tendency to pneumonia. In some cases this latter can be demonstrated; in others it can only be surmised.

In elderly persons there is usually some chronic visceral disease which renders the bronchitis something more than a mere cold when looked at from a prognostic standpoint. I now have a case in mind. I was asked to see in consultation a lady of sixty-nine who was having a bronchitis with moderate elevation of temperature and whose rapid feeble pulse was giving the attending physician considerable uneasiness. The patient was in a really serious condition; rectal temperature 102.4° F., respirations 48, pulse 140. The lungs were resonant excepting slight impairment at the two posterior bases; the chest was absolutely filled with whistling and fine bubbling râles which entirely disguised the character of the breath-sounds; the heart was plainly in a state of chronic myocarditis with a systolic apex murmur. The patient's condition had developed insidiously upon a cough of several weeks' duration.

I believe the condition was a broncho-pneumonia of influenzal origin because the signs were those of bronchitis that had invaded the bronchioles. It illustrates how readily an originally acute bronchitis may run on into a capillary bronchitis in an old person with cardiac disease and feeble resisting power.

Physical Signs. Inspection.—Simple acute bronchitis in a healthy adult occasions no conspicuous alteration of respiratory movements. In very young children and in the aged or feeble, breathing is likely to be accelerated and, if the implication of the smaller tubes be pronounced, there may be noticeable dyspnœa and even cyanosis. When however marked respiratory embarrassment is present it probably indicates some added condition as pneumonia.

Palpation.—This generally proves negative but in infants or persons with elastic chest-walls there may sometimes be felt the so-called rhonchal fremitus or the vibration produced by the passage of air during inspiration through the liquid secretions filling the tubes. Such a fremitus usually disappears after cough.

Percussion.—Pulmonary resonance is usually preserved, but it may be impaired at the bases behind when the bronchioles are filled with mucus and lobular collapse results. This impairment clears up after cough has emptied the tubes. In very young children cracked-pot resonance may be obtained in front if percussion is heavy and in particular if the infant is crying.

Auscultation.—This is likely to furnish information of real value. In acute specific bronchitis, as in typhoid fever, there are moist and dry râles of variable number and distinctness, generally most apparent at the bases of the lungs. In simple tracheobronchitis râles are not generally audible, but instead the breath-sounds are partially suppressed and more harsh than normal. As the disease passes from the stage of congestion into that of free secretion the ear may detect rhonchi of varying pitch and intensity or coarse mucous râles, all of which change or disappear after cough. The breath-sounds are never bronchial but they may be exaggerated and in some instances nearly or quite obscured by the râles.

Diagnosis.—In acute bronchitis accompanying the specific fevers and the diseases of childhood diagnosis may generally be made by the results of chest examination; but in simple tracheobronchitis reliance must be placed upon history and symptoms rather than upon positive physical findings. Indeed it may be said that the real value of a chest examination is to be found in the negativeness of the results in the great majority of instances, particularly in robust young adults.

Differential Diagnosis.—This affection is to be differentiated from pneumonia of whatever origin and nature and from incipient pulmonary tuberculosis. As regards the various forms of pneumonia it may be stated in general that the detection of an area of dullness with bronchial breathing and the usual restriction of the findings to one lung speak for something more than bronchitis. This latter attends the former affection but is to be considered as only a subordinate condition. Other distinguishing features are given in the appropriate chapters.

One so frequently hears of incipient tuberculosis being pronounced acute bronchitis and such an error is so unfortunate to the patient that no case with a history of recent cough with or without expectoration should be dismissed without a careful determination of its real nature. The details of differential diagnosis cannot be given here since they are to be found in the chapter dealing with the diagnosis of pulmonary tuberculosis. But let it here be emphasized that it is a mistake to restrict an examination to the front of the chest since in tuberculous disease the early signs are often confined to the back and are almost always unilateral. In every doubtful case careful and often-repeated search for the bacilli must be made. It is far better to be too suspicious concerning the tuberculous nature of a bronchitis than too decided in the other direction.

Prognosis.—In acute specific bronchitis the prognosis may be said to be that of the general infection, since with recovery from the latter the former disappears. In very young children, however, the bronchitis itself may seriously compromise the prospect of recovery. If in any such patient the respiration becomes considerably embarrassed and cyanosis appears or the râles point to implication of the finer tubes the case must be looked upon with apprehension, since catarrhal pneumonia has probably been added to the bronchitis or atelectasis is seriously restricting respiratory capacity.

On the other hand an acute tracheobronchitis in a previously healthy young adult rarely proves a serious matter. If properly managed it may be expected to terminate in recovery in the course of two or three weeks. A bronchitis complicating chronic disease of heart, kidneys, blood-vessels or lungs may on the contrary present a very grave prognosis. So long as it is restricted to the larger bronchi it is not dangerous, but should it extend to the finer ramifications there is always a liability to pneumonia.

In the aged in particular an acute bronchitis should never be lightly regarded, not only because of its possible termination in catarrhal pneumonia but because it many times attends and conceals a patch of fibrinous or influenza pneumonia. The appearance of drowsiness or of prostration out of proportion to the apparent gravity and extent of the bronchitis should always excite alarm.

CHAPTER III

CHRONIC BRONCHITIS

CASES of chronic bronchial catarrh, like the acute, are divisib
main categories; viz. (1) those that are due to some specific pathog
ism, (2) those that cannot be regarded as a persistence of a specifi
but that result from frequent repetitions of an ordinary trache
or that develop insidiously and may therefore be considered as a
inflammation of the air-tubes. Accordingly, these two varieties
scribed separately, so far as concerns their symptomatology.

Symptoms of Chronic Specific Bronchitis.—Cases of this
out of an acute influenza bronchitis more frequently, as it seems
out of any other infectious disease. Measles and whooping-cough
may lead to a persisting bronchial catarrh, but such is not the
means. Badly nourished children living in an unhygienic envir
more likely to develop subacute broncho-pneumonia or tuberculos
on the other hand are more likely to lay the foundation for th
cough and expectoration in a previous attack of *grippe* or even pn
pneumonia.

They rally from the acute infection and lose all symptoms, e. g
then before their cough has wholly disappeared they return to busi
fancy they can throw off the cough in time and hence they drag
about feeling weak and miserable but not actually ill. They
or less expectoration of a mucous or muco-purulent kind and
even wheeze somewhat on extra exertion or after coughing.
hearty appetite, perspire unduly and often present a too frequent
pulse.

In this state they at length consult a physician who finds eith
tion of temperature or at most but a trifling one, of 99° F. or possi
in the evening. Examination of the chest discloses at one posterio
impairment of resonance and a variable number of fine or m
mucous râles which nearly or quite obscure the breath-sounds.
is examined and found to contain streptococci or staphylococci wi
bacilli or pneumococci in such numbers that doubt cannot be en
their coming from the lungs and not the mouth.

Such cases may clear up on general tonics and expectorant
most part are likely to drag on for months or eventuate in pneum
the patients are sent to another climate where they can live out of
such a climatic treatment they usually recover rapidly and perma

Symptoms of Chronic Nonspecific Bronchitis.—This form of chronic bronchitis usually develops out of the acute. A simple tracheo-bronchial inflammation is contracted in the Fall of the year and runs the ordinary course and after two or three weeks has just about disappeared when, from fresh exposure or some other unknown determining cause, an acute catarrh is again lighted up. In this way one attack is succeeded by another, until at last the patient finds himself with a persistent cough which shows little if any tendency to improvement as weeks go on. The chronic malady has in reality grown out of repeated exacerbations of an originally acute catarrh.

In another set of cases the acute bronchitis manifests no tendency to recovery after the usual length of time but persists with a degree of obstinacy that carries it well on into a period of weeks before the patient comes to realize its seriousness. It is not always quite clear how soon the affection is to be looked upon as chronic. Gintrack sets forty days as the length of time after which a bronchitis may be reasonably said to have become chronic (Hofmann). This is certainly conservative since an ordinary attack of acute tracheobronchitis may terminate in recovery in from two to three weeks.

In the former group of cases may be found healthy young adults whose occupations predispose them to vicissitudes of weather or who because of their good health think they can easily throw off their colds and commit such foolish indiscretions as going from a heated ballroom into the outer air to cool off.

On the other hand it is likely to be the person of feeble resisting power in whom the bronchitis tends to persist without appreciable attempt at resolution. Such an individual may have stiff vessels or chronic cardiac or renal disease, or, without any demonstrable lesions, he may be of a gouty habit and have mucous membranes that for years have shown more or less irritation whenever his elimination became defective. Very commonly, therefore, it is the elderly and infirm or the aged, who thus develop a chronic out of an acute bronchitis.

The symptoms of chronic bronchial catarrh are *cough* and *expectoration*. In fact whatever the variety of the disease or whatever its mode of development, these are the leading features. Other symptoms that subsequently appear are manifestations of the effect of the bronchial affection on the general health or on other organs, chiefly the lungs and the heart.

In otherwise healthy individuals with chronic tracheo-bronchial catarrh the general health does not suffer and the only inconvenience they experience is from the *cough*. This, as a rule, is most troublesome in the early morning, either before they arise or immediately thereafter. By 5 or 6 o'clock they are aroused by a tickling or uneasiness in the lower part of the throat or behind the upper end of the sternum which impels them to cough. Having once yielded to such desire they find it practically impossible to stop it for more than a few minutes at a time until they have become fully aroused and sleep is no longer possible. In other cases the main stress of coughing comes when they leave their warm beds and begin dressing, in consequence probably of the temporarily increased bronchial congestion caused by the chilling of the skin. Still other patients do not cough until they drink something hot at breakfast or inhale cold air as they leave home for places of business.

This symptom also varies much in severity, depending apparently upon the facility and amount of the expectoration. When this is thin and of considerable quantity it may be expelled by a few energetic efforts and then not reaccumulating quickly may leave the patient in comfort for the rest of the day. In most cases, however, the sputa are not so easily expelled and the morning cough is prolonged for many minutes. Occasionally persons are compelled to keep up a violent even retching cough until quite exhausted or very much out of breath and red in the face.

The *expectoration* in this form of chronic tracheobronchitis varies in both character and amount. It may be scanty and viscid or abundant and diffluent. When scanty and viscid it usually consists of pellets or roundish masses of mucus which are brought up with difficulty. When the sputum is more fluid and abundant it presents a whitish or yellowish appearance and is termed muco-purulent or purulent according to the preponderance of mucin or leucocytes. It may in some cases have a greenish or greenish-yellow color.

In cities or in localities where the atmosphere is laden with dust and smoke the expectoration usually takes on a dirty gray or even blackish hue. It is rare for the sputum in simple chronic bronchitis to be streaked with blood, but when so streaked, the blood is extravasated in consequence of the violence of the coughing efforts. Hofmann states that vomiting does not attend the act of expectoration in simple chronic bronchial catarrh and that when emesis occurs it is in phthisis and results from the serious nutritional disturbance that has taken place. When, nevertheless, strings of tough mucus stick in the glottis, as they not infrequently do, they cause a degree of retching which if it does not actually terminate in emesis proves very straining and fatiguing. The more copious the sputum, the more easily it is expectorated because rendered diffluent by the transudation of serum.

Examined microscopically the sputa are seen to consist mainly of leucocytes with here and there alveolar epithelium, and considerable numbers of cylindrical epithelium. In some instances eosinophile cells may be discovered (Gollasch, Teichmüller) and when present they are usually found in the sago-like pellets of tenacious mucin. In occasional instances these cells may be so numerous that as suggested by Teichmüller, the catarrh may be spoken of as an eosinophilic bronchitis (Fraenkel). Bacteria are also present in the sputa, especially in the muco-purulent or purulent expectoration and then consist of the multitude of organisms which abound in the mouth and upper portion of the air-tubes. They are a secondary admixture but in my opinion exert considerable influence in the maintenance of the bronchitis.

In most instances of this chronic bronchial catarrh now described, the clearing out of the tubes in the early morning is succeeded by some hours of immunity from cough, or at the most, the patient gives but an occasional hack or two, without expectoration.

In some instances, perhaps in most, so long as the disease is mild, there are no more severe paroxysms of coughing until the next morning. Other persons find more or less impulse to the act upon going to bed and resuming the recumbent position, but at length fall asleep and remain undisturbed for the remainder of the night. The freedom from evening cough probably bears a direct relation to the rapidity and amount with which secretions collect. If they are scanty they do not accumulate in sufficient quantity to occasion discomfort until toward morning. When on the contrary they form in considerable amount during the latter part of the day, yet have not been expectorated, the assumption of the dorsal decubitus causes the mucus to gravitate to the junction of the main bronchi with the trachea or to the posterior wall of the latter where the great sensitiveness of the membrane leads to reflex irritation and cough.

If patients in this stage of chronic bronchitis are examined they are generally found in good physical condition and declare they are well with the one exception of their bronchial catarrh. Examination of the chest is also negative, the percussion note being everywhere clear and the respiratory sounds free from râles.

The state of the patient remains as described during the Winter, being somewhat intensified by bad weather and ameliorated whenever there comes a spell of warm sunshiny days. Accordingly, as Spring advances and the proportion of pleasant weather increases the bronchitis shows corresponding improvement. When at length Summer arrives and the weather becomes settled and warm, cough disappears and the patient congratulates himself on having gotten rid of his troublesome symptoms.

Such is the history of the beginning of the mildest type of chronic bronchitis which, because confined wholly or chiefly to the winter months, has been termed *winter cough*. It is possible for an individual who has thus suffered for a single season to escape a return of his malady the succeeding Winter if he take extra good care of himself or spend the second Winter in some mild climate. In most cases, however, persons are not able to adopt such measures and, flattering themselves that they have lost their old trouble for good, they are careless when the next Fall comes around. Consequently they again contract a bronchitis and thenceforward, though probably in an exaggerated degree, they experience the same series of events as the year before. The second Summer again brings them immunity from their complaint on the whole, but they perceive a sensitiveness to changes of temperature even in Summer, which is new to them. The third Winter brings back the winter cough even more promptly and emphatically than before.

Thus with each successive year their enemy returns and with each return his attacks become more aggressive and more difficult to repel. In this fashion years pass until, at length, the general health shows the effects of the chronic disease. Of course, the rapidity with which the bronchitis undermines the health and the degree of this ill effect depend upon other factors than the local disease itself.

If the previous state of health, the occupation, habits and general environment are such as make for good resisting power, the inroads of the bronchial affection may be insignificant for many years. Thus I know a man who has had a winter cough for a number of years, but who yet manifests no shortness of breath and appears to suffer no inconvenience aside from that of his annually recurring cough.

In the majority of cases, on the contrary, chronic bronchitis develops in persons, e. g. laborers, laundresses, machinists, etc., whose habits and environment are not of the best, and hence after a very few seasons it seriously undermines their general health. They at first find their breath growing a little short, especially after coughing, and as this act has become frequent its effect upon their wind is serious. Next they find they get out of breath in walking a little hurriedly, or in the face of a strong wind. Year by year their breathlessness increases until, at length, they are no longer able to perform their work.

If now the chest is examined it is found that emphysema has developed and the right ventricle is hypertrophied, while the peripheral arteries are very likely to feel stiff.

Even before the lungs and heart exhibit this degree of secondary effects, the nutrition in general has been influenced injuriously. The weight declines, the strength diminishes and the color grows paler than normal. Appetite fails or the ingestion of food is seriously interfered with by the cough and expectoration which it excites. The bowels are usually constipated, and bloat up after meals. The urine is scanty and loaded with urates, or it may be of low specific gravity, may flow more freely at night, and may now and then show a trace of albumen and casts.

The chest is now filled with râles and after coughing the patient wheezes distinctly. Attacks of intensified dyspnœa come on which, because paroxysmal, are designated asthmatic. They are indeed what is known as *bronchitic asthma* and in this advanced stage bear an intimate relation to the emphysema. The patient's condition is now truly pitiable. Incapacitated for work or even for the ordinary enjoyments of life he sits at home by the fire, racked by frequent fits of coughing, suffering from headache or myalgia due to the violence of his tussive efforts, panting and unable to talk without intensifying his sufferings. Thus year after year goes by and at last a burden to himself, he finds relief from his misery in death which comes as a result either of an attack of acute bronchitis or pneumonia, or of cardiac inadequacy with all its phenomena of stasis.

Fever is not an essential symptom of chronic bronchitis and is for the most part entirely wanting. When it does occur, it is usually due to an acute exacerbation of the bronchial inflammation or to some intercurrent affection. In cases which have led to bronchiectases with retention of the secretions, or which without bronchial dilatation display sputa of a fœtid character, (putrid bronchitis) pyrexia is not uncommon. It is then probably a manifestation of intoxication from absorption of products of decomposition and is of the remittent type. Some elevation of temperature is not unusual in the last few weeks or days of life and may be the main indication of a terminal pneumonia. The occurrence of fever, therefore, at any time, should be regarded with uneasiness and calls for investigation.

Pain is likewise not a legitimate feature of this disease. When present it is generally either a myalgia or an intercostal neuralgia. The former usually comes from the strain of violent coughing and is then very apt to be felt in the epigastric region at the point of insertion of the diaphragm. Fothergill, himself a sufferer from chronic bronchitis, describes a sudden intense pain in the lower part of the thorax, at either side, which he attributes to the tearing loose from its attachment of a fibril of muscle and which necessitates firm pressure with the hand on the painful spot during subsequent fits of coughing.

Occasionally pain in the side of the chest may be so sharp and occasion such a catch in the breath as strongly to suggest pleurisy, but is differentiated from that of the latter by the absence of friction-sounds. Some of the neuralgias felt in the upper zone of the chest, generally in the second and third left interspaces being symptomatic of visceral disease, as pointed out by Head, are very persistent. Congestive headaches may also be produced by prolonged paroxysms of coughing and in some individuals are very distressing.

Dyspnœa.—In the early stage of chronic bronchitis shortness of breath is not a prominent symptom. A prolonged fit of coughing may take away the breath for a few moments immediately following, but so soon as it has been recovered from the patient experiences no further respiratory embarrassment until another paroxysm of cough ensues. As the disease progresses, however, breathlessness on exertion gradually develops and in the last stages may become very distressing. The cause of this dyspnœa is partly pulmonary and partly cardiac.

If the disease invades the smaller tubes, ingress of air may be impeded by swelling of the mucosa and collection of secretions within the bronchioles. In most of the advanced cases the difficulty of respiration is in chief measure owing to the secondary emphysema and the immobility of the lungs which results. When this state of things has been produced, it is common for the dyspnœa to assume the asthmatic type. In other cases there seems to be a tendency for the bronchitis to display asthmatic paroxysms from the beginning or soon thereafter. In some, the asthmatic paroxysms are so marked as to merit the term *bronchitic asthma*.

As already stated another cause of breathlessness lies in the circulatory disturbances secondary to the bronchial catarrh. The mechanical impediment to pulmonary circulation effected by the emphysema, as well as the lessened driving power of the dilated right ventricle, occasion dyspnœa of effort the same as in primary cardiac incompetence and they are in part responsible for shortness of breath by interfering with adequate oxygenation of the blood. When all these causes have combined in the terminal weeks of life and the expansion of the lungs has become still further restricted by hydrothorax and perhaps ascites, the difficulty of breathing is likely to amount to orthopnœa. This prevents the rest to the weary body obtainable in the recumbent posture and greatly intensifies the patient's distress from other causes.

Digestive disorders do not belong properly to the history of chronic bronchial catarrh in its early stages. If present at all they are an accidental accompaniment or together with the bronchitis are secondary to some other primary affection, as cardiac or renal disease. When on the other hand secondary pulmonary and circulatory effects have been produced, digestive disorders become very common. They consist chiefly of fermentative indigestion with consequent flatulent distention of the stomach and bowels. Appetite is more or less impaired, and if an attempt is made to eat, the patient quickly feels full and uncomfortable or is annoyed by eructations of gas. The distention of the stomach occasions upward pressure on the diaphragm and intensifies the shortness of breath.

If actual pain is experienced in the epigastric region, this is due not to the indigestion but to hepatic stasis or the strain of coughing as has been already stated. Constipation is usually added to the bloating that follows the taking of food and is owing largely to the flatulence. In some cases diarrhœa alternates with constipation. The conjunctivæ are apt to have a sub-icteric tint and the general aspect may betoken defective nourishment. Some chronic bronchitics now lose weight while others retain their weight but are flabby and have protruding tympanitic bellies.

The *urine* is apt to be deficient in amount and deposits urates upon cooling. Albumen does not appear until in the last stages of the bronchitis and is then due to renal congestion. If chronic nephritis complicates or is responsible for the bronchial catarrh, the urine presents the changes characteristic of the renal disease. Women not infrequently complain of their inability to retain their urine in consequence of the strain of coughing.

Excepting *headache* already mentioned as due to congestion, and perhaps also *irritability of temper* or *impairment of memory*, likewise due to faulty cerebral circulation, there are no special mental symptoms. In the terminal stage of the disease mild *delirium* may set in and is then probably the result of an autotoxæmia. There may be such a degree of carbonic acid poisoning as to cause *stupor*. In this state of circulatory embarrassment leading to headache and orthopnœa, *sleep* is likely to be fitful and disturbed by multifarious dreams which rob it of all refreshment.

Circulatory Disturbance.—The heart becomes seriously affected in nearly all cases of chronic bronchitis, especially when this latter has led to secondary emphysema. The right ventricle undergoes hypertrophy and subsequently dilatation, while the myocardium suffers more or less degeneration. These changes are due partly to mechanical interference with the circulation through the lungs and partly to sclerosis of the coronary vessels and consequent diminution of cardiac nutrition.

Hypertrophy of the right ventricle does not develop early in the course of chronic bronchitis unless as a consequence of associated emphysema or of sclerosis of the pulmonary artery, which, as Romberg states, often precedes the development of the bronchial catarrh. Ordinarily the hypertrophy comes on after several years, and then because of vesicular emphysema or fibrosis or possibly in consequence of the chronic cough and rigidity of the lungs. It is therefore a manifestation of the strain to which the ventricle has been subjected.

An additional factor is the heightened blood-pressure in the pulmonary system which is secondary to increased pressure within the left ventricle, which latter pressure results from sclerosis and hypertension in the general arterial system. We have no difficulty, therefore, in understanding the mode of production of the hypertrophy of the heart seen in these cases.

Such compensatory increase of its wall endures for years, but as it possesses but a limited amount of reserve power there is a limit to its hypertrophy. It intense pain in the lower part of the thorax, at either side, which he attributes to the tearing loose from its attachment of a fibril of muscle and which necessitates firm pressure with the hand on the painful spot during subsequent fits of coughing.

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Such compensatory increase of its wall endures for years, but as it possesses but a limited amount of reserve power there is a limit to its hypertrophy. It at length becomes overtaxed either by the increase of its labors or through some sudden strain and its cavity begins to dilate. The dilatation now acts injuriously upon cardiac nutrition, even if this has not suffered somewhat before from other causes.

The cardiac veins empty into the right auricle and if blood-pressure in this chamber is raised, as is the case whenever blood-flow through the lungs is interfered with, there is corresponding impediment to outflow from the coronary veins. The ultimate result of such stasis is impaired nutrition of the cardiac muscle in general, but of the right ventricle in particular because of its augmented work. Not only are the products of its metabolism not adequately removed, but its aeration is defective and degeneration results. In all these ways therefore the heart suffers in its integrity.

Hypertrophy of the right ventricle is shown by epigastric pulsation and the ringing quality of the pulmonic second sound. As dilatation ensues, epigastric pulsation grows less powerful and cardiac dullness increases to the right, when not obscured by pulmonary emphysema.

The brunt of the heart-strain being borne by the right ventricle temporary dilatation may supervene whenever a chronic bronchitis is intensified by an acute exacerbation. The wall of the right ventricle is relatively thin and yielding, so that dilatation is easily brought about and may be readily recovered from. It is a matter of familiar observation therefore in chronic bronchitis that a systolic murmur develops in the tricuspid area during periods of extra stress and disappears with the subsidence of the acute bronchitis or other condition which has temporarily increased the strain.

With more lasting strain or a greater degree of distention other and more reliable signs of relative tricuspid incompetence appear, and perhaps remain to the end of the case. We are wont to regard this occurrence of tricuspid regurgitation as a sign of danger, and so in a sense it is, for it indicates a degree of strain too great for the ventricle's resistance. This tricuspid insufficiency, however, acts as a safety-valve provision: it serves to protect the ventricle from fatal overdistention in cases of chronic bronchitis and emphysema just as much as in acute overstrain in athletes who are enduring prodigious physical effort.

With the establishment of regurgitation, strain is lifted somewhat from the greatly distended ventricle and thrown upon the auricle and great veins, thus for a time relieving the former chamber of what might, if it continued, prove a disastrous degree of dilatation. This conservative process is therefore not a sign of danger in itself but a measure of the degree of strain to which the ventricle is exposed. In reality I believe it serves to prolong life. It adds fresh discomfort to the patient, causing hepatic engorgement, congestion of the other abdominal viscera and perhaps increasing the tendency to anasarca, but it gives the sufferer a respite from cardiac paralysis.

There is still another direction than that already considered in which increase of blood-pressure in the right heart proves disastrous to the sufferer from chronic bronchitis; this is pointed out by Samuel West. The contents of the bronchial veins is emptied in two directions: (1) through the superior intercostal veins and vena azygos into the general venous current and thus to the right auricle, (2) into the pulmonary veins by means of anastomoses. So soon therefore as blood-pressure is raised in the pulmonary veins it retards the outflow from the bronchial venules, increases congestion within them and augments the swelling of the bronchial mucosa.

When at length tricuspid insufficiency leads to back pressure in the right auricle, the blood is dammed back into the systemic veins that receive the major portion of the contents of the bronchial veins, and the congestion of the bronchial mucous membrane is still further increased. The entrance of air into the lungs and the oxygenation of the blood become still more difficult, and nutrition of the heart-muscle suffers proportionately. Thus, as West says, a vicious circle is set up, the bronchitis tending to injure the heart and this in turn to aggravate the bronchitis. It is therefore easy to understand the orthopnœa, cough and sero-mucous, even bloody, expectoration on the one hand, and the general venous stasis and feeble heart-action on the other, which torment many a sufferer from chronic bronchitis in his last struggle for life.

Varieties of Chronic Bronchitis. The Asthmatic Form.—The occurrence of paroxysmal intensification of dyspnœa, the so-called bronchitic asthma, has been previously mentioned in considering dyspnœa. It is especially frequent in long-standing cases that have led to emphysema and is very apt to be treated as ordinary asthma, the physician failing to recognize its relation to an antecedent chronic bronchial catarrh. Such attacks are held by some authors to be due to bronchial spasm, and the relief sometimes afforded by antispasmodic remedies appears to support such a view. It has seemed to me that in some instances the attacks might be caused by temporary increase of bronchial hyperæmia like the fluxionary hyperæmia suggested by Weber for some cases of true asthma. It is also not impossible that in some cases the attacks are in reality instances of cardiac asthma so-called and due to temporary increase of heart-weakness.

In contrast to the foregoing, one occasionally sees cases in which the ever-recurring attacks of bronchitis are invariably attended by asthmatic breathing; and the frequency of such attacks seems to indicate that the bronchial mucosa is never quite free from a certain degree of inflammation although cough and other symptoms are too insignificant in the interim to attract attention.

I have recently seen such a case in a young woman of twenty who declared she had suffered from cough and asthma for only a year. Her statement was that she had asthma only when she took cold. On close inquiry it was ascertained that she was never entirely free from cough, although she gave no heed to it when not suffering from asthmatic attacks, and that she had "wheezed," especially on exertion, for two or three years.

Her father had been asthmatic but had died from Bright's disease at sixty-seven. One sister had suffered from "wheezing" for a short time but had been cured. The patient in question had not suffered from her asthma before she began to work in a nail factory three years ago. The nature of the occupation suggested the possibility of metallic dust as the ætiological factor; but inquiry failed to substantiate the certainty of the inhalation of dust, for although her particular work was the polishing of horseshoe nails with a machine she was not conscious of very much dust in the process.

Examination revealed a slightly anæmic-looking but not emaciated girl whose chest was not deformed and whose glandular system was negative. She was not a mouth-breather and, excepting when she had taken cold, could breathe easily through her nose. The chest was resonant, but over both lungs the respiratory murmur was obscured by squeaking and groaning rhonchi during both acts but especially with expiration. Her temperature was normal and the heart-findings were negative with exception of marked intensification of the pulmonic second tone. There were no digestive or menstrual disorders that could be elicited.

The case seemed to be quite clearly one of asthmatic bronchitis, and yet, lest it might have a tuberculous basis the young woman was instructed to send her sputum for examination. She said her cough was so tight that she did not know if she could collect any, but would do so if possible. The remedies ordered were syrup of hydriodic acid 1 drachm four times each day and also 5 grains of terpine hydrate the same number of times daily. Some purulent sputum was received three days thereafter, which aside from pus cocci and mucus contained no other organisms. One week later she was seen again and then stated that she was quite well. Nevertheless a few sibilant râles were detected at the left posterior base, apparently the only remains of extensive bronchitis of a week previous.

The following case, it seems to me, may also be classed as one of asthmatic bronchitis, although the findings in the left upper lobe give a suspicion of a tuberculous foundation. The patient is a female, a school-teacher aged twenty-nine years who was first seen by me at her home in DeKalb, Illinois, in the Spring of 1901. She had been confined to the house for a few weeks with what she called a cold on her chest, and asthma.

Family history was free from tuberculous taint or other pulmonary disorders. It was stated that she had coughed more or less for three years but that tubercle bacilli had not been discovered in her sputa. During the Summer of 1900 she had spent a few weeks in Colorado with much benefit.

The patient was in good flesh having lost only a few pounds in weight and at the time of examination weighed one hundred and sixty pounds, her height being five feet eight inches. Inspection was negative as regarded the chest, but it was noted that her respirations were somewhat difficult. Percussion disclosed moderate dullness of the left upper lobe to the third rib in front, and behind nearly to the inferior angle of the scapula. Upon auscultation the breath-sounds could not be distinctly heard because of the multitude and intensity of râles which were mainly sibilant, although in front, beneath the left clavicle there were also dry crackling râles. With exception of a few rhonchi over the main bronchi in front, the right lung appeared free from physical signs.

From the history, the dullness at the left apex, and the unilateral location of the bronchitis the condition was believed to be tuberculous, notwithstanding the absence of tubercle bacilli, and the advice was given to go to Denver without delay.

She remained there a year and after a time lost all symptoms; the bronchitis cleared up, although slight dullness still persisted at the apex. Repeated search was made for tubercle germs but always without success.

In August of 1902 this patient returned to her home in Illinois. Being imprudent she soon thereafter contracted a cold and again suffered from difficulty of breathing of an asthmatic type and from cough without expectoration. In October she consulted me and I found a condition of things very similar to that of March, 1901.

Her breathing was labored and wheezy, the left upper lobe was dull, and throughout the left lung were innumerable sonorous and sibilant rhonchi, as well as dry crackling râles in the upper part. There were also some rhonchi in the right lung behind, but whether they originated there or were transmitted from the opposite side I could not determine. Her temperature was strictly normal, she had not lost weight or strength and tubercle bacilli could not be found in the small amount of sputum she was able to send me a few days subsequently.

She was put upon syrup of hydriodic acid and terpine hydrate in large doses (30 grains daily) with the result that in a week she had lost her asthma and very largely her cough.

Careful examination now disclosed improved resonance over the left upper lobe and an entire absence of all râles with exception of a few whistles at the posterior base. It seemed apparent that a part of the dullness in the apex had been owing to clogging of the bronchioles by secretions.

A week or two later this patient suffered exposure in wet weather and had a relapse, shown by asthmatic breathing and increased cough but not by such numerous or extensive râles as before. In December, 1902, she was entirely free from symptoms excepting a moderate degree of cough and her chest contained no râles. Moderate dullness was present at the left apex, but careful search failed to disclose signs of cavity. I therefore believe that the process was one of fibrosis, whether of tuberculous origin I am not certain although this seems probable, and that this condition predisposed her to bronchitis. This displayed a marked tendency to chronicity and was always of an asthmatic type. Although very suspicious of the exact nature of this case I nevertheless think myself justified in treating it as a bronchitis and in classifying it as belonging to the asthmatic variety.

Mr. R., student, aged twenty-three, sought advice because of cough, difficult expectoration and dyspnœa. His mother died of pulmonary tuberculosis, but otherwise his family history is unimportant. He was a fat healthy baby until his third year when he took cold, and since that time he has had a chronic cough, although in the Summer it has not given him special trouble. He thinks he was a mouth-breather as a child. From his sixth or seventh year on, he has been subject to occasional attacks of asthma. A year ago a spur was removed from his sæptum and since that time he has been comparatively free from his asthmatic attacks, although he has had his cough when the weather was inclement.

A month ago he was lying on the sofa when some of his companions began to tickle him which led to his both laughing and struggling to get up. He suddenly became seized with a violent paroxysm of dyspnœa which caused him to tug for breath and he grew cyanosed. This attack did not abate for nearly a week and during some of that time he was obliged to inhale oxygen.

His cough and dyspnœa have since persisted. The former has not been severe and at the date of my examination seemed to come on only, as he expressed it, "when he had to clear out his lungs." The sputum was usually in the form of clear soft balls that were single or several adhering together. His pulse was 110, small and weak but regular. His temperature was 99.2° F. and his difficulty of respiration was shown by deepened inspiratory efforts rather than by a notable hastening of the act.

Examination of patient: Height five feet eleven inches, weight 135 pounds. Inspection showed his chest long, slender, and very short in its antero-posterior diameter at the upper part, but relatively deeper at the base. When the shoulders were thrown back, the scapulæ met and the vertebral column was thrown forward so as to encroach still further upon the anterior mediastinum. There was marked pulsation in the region of the xyphoid cartilage and around about, with striking depression of the adjacent costal cartilages.

The apex beat could not be definitely located. The percussion note was resonant throughout, reaching in the right nipple line to the upper margin of the seventh costal cartilage, and the excursion movement of the diaphragm was preserved. Over the front of the chest, particularly over the bifurcation of the trachea and the right main bronchus, were loud musical rhonchi heard chiefly on expiration but also during a forcible inspiration. The hand laid on this part perceived a distinct fremitus synchronous with the rhonchi and disappearing when the breath was held. Similar râles were audible posteriorly and were brought out very beautifully by more vigorous respiration.

These râles were of varying quality, at one instant cooing, at another rumbling and the next instant perhaps changing from a low to a high pitch and following by a short interval the beginning of the expiratory act. They did not shift their location, but varied in intensity in the same locality. Moist râles were nowhere detected.

The only feature worthy of note in the examination of the heart was found in its abnormally low position shown by both palpatory and plessimetric percussion. The superior boundary of relative cardiac dullness was discovered to be in the third interspace at either side of the sternum close to the upper border of the fourth, instead of the third costal cartilage. The inferior boundary was correspondingly too low, being at the junction of the sixth and seventh costal cartilages. Both at the right and left the boundary of deep cardiac dullness was normal. The sounds were clear and free from murmurs, but the pulmonic second seemed relatively a little diminished.

The case seemed plainly one of chronic bronchitis with an asthmatic tendency and slight secondary emphysema and cardioptosis. From the history and shape of the chest it was concluded that nasal obstruction had been responsible for the defective thoracic development and the deformity at the anterior base. The lowered position of the heart was also attributed to the contour and shallowness of the chest at its upper part, which did not furnish room enough for the base of the heart, and forced it downward into the relatively more capacious lower mediastinum.

A year later this supposition was found to be erroneous. The patient had been in Colorado for months and was no longer asthmatic. The diaphragm had returned to its normal position carrying the heart upward as shown by its superior boundary being at the upper border of the third instead of the fourth costal cartilage. Consequently it seemed clear that the low position of the heart a year previous had been due to the depression of the diaphragm which, although its excursion movements were not abolished, had assumed, more or less permanently, the position it occupies during inspiration. This of course was owing to the emphysema occasioned by the asthma. It may be added that the patient was entirely free from his bronchitis. In this case the history of chronic cough since childhood led me to conclude that a bronchitis was the primary while the asthmatic manifestations were the secondary condition.

Dry Bronchitis.—This variety known as *bronchitis sicca* was originally described by Laennec as *catarrhe sec.* Authors differ in their statements regarding its frequency. There is no doubt however that in old or gouty subjects there is commonly encountered a form of chronic cough which is characterized by the violence and frequency of the cough with disproportionately little result in the way of expectoration.

The paroxysms are in some instances of great severity, the patients becoming red or even purple in the face, very much blown and exhausted. If sputum results, it is but little more than a pellet of clear viscid mucus, with or without frothy serum. The patients suffer more or less from dyspnœa, due to emphysema and deteriorate in general health. Physical signs within the chest are few, if any, consisting chiefly of sibilant râles at the bases behind. The affection is very refractory to treatment.

I recall a case of this kind in an old German whom I treated in 1884. He was a tall, gaunt, large-framed man with well-marked evidence of arteriosclerosis and the attendant changes in the heart and kidneys. His great complaint was hard dry cough, with very scanty difficult sputum, and some shortness of breath. The lungs were negative as regarded signs of chronic bronchitis, but it was apparent from the state of the circulation that there was bronchial congestion. Various expectorants and cardiac tonics were tried, all to no purpose. At length it was determined to try the effect of a powerful cathartic, and accordingly he was given five grains each of calomel and jalap. The old man did not return for three months. He then came for another powder, declaring he had never had anything do him so much good in his life. The benefit was due to the lessening of bronchial congestion.

Osler describes a variety of dry bronchitis which he has observed in women and which is characterized by obstinate cough and a small amount of expectoration. The cough usually makes its appearance between the ages of twenty and thirty and persists "without serious impairment of the health." There are no discoverable changes in the respiratory organs to account for the symptoms.

Bronchorrhœa serosa of Biermer and chronic pituitary catarrh (Laennec) are terms given to a variety of bronchitis which is characterized by such profuseness of expectoration as to constitute a veritable bronchial flux. The disease is seen most frequently in aged people or such as are debilitated and prematurely old. Thus one of Andral's cases was in a man only forty years of age but who was considerably emaciated and reduced in strength.

There is nothing in the examination of the chest to distinguish these cases from others of chronic bronchial catarrh and therefore the characteristic feature is the expectoration of large amounts of serous sputum, as much as one or two pints in twenty-four hours. The sputa are variously described as clear or frothy fluid containing flakes or strings of mucus, or as looking like a solution of gum (Andral), on the surface of which float roundish masses of greenish-yellow mucus. The sputa possess no fœtor and are expectorated easily, coming up in mouthfuls, or in some instances as if vomited. Expectoration usually occurs in the early morning and again in the evening but may be repeated at intervals during the day.

Patients appear to succumb to the exhausting effects of their disease after a course of months or a few years, although in one of Laennec's cases the affection lasted many years. Autopsy is said to reveal nothing more than the ordinary changes of chronic bronchitis.

The diagnosis of bronchorrhœa is based not on physical signs, since, as in Andral's cases, examination discloses nothing more than the usual findings of chronic bronchial catarrh, but upon the copiousness and character of the sputa. Care should be taken not to confound these with cases of acute pulmonary œdema or with the copious serous expectoration that is said sometimes to follow paracentesis thoracis. According to Gee this latter discharge looks like that of bronchorrhœa serosa but differs from it by containing albumen instead of mucus, as determined by chemical analysis. The disease might also be confounded with bronchiectasis in which large amounts of sputum are evacuated at intervals, but in the latter the expectoration is generally offensive and there may be the signs of bronchial dilatation.

Treatment is unavailing but should consist chiefly of measures calculated to build up the general health. It has not been my good fortune to see a case which I thought could properly be called one of bronchorrhœa.

Putrid bronchitis, also termed fœtid or septic bronchitis, is distinguished from other forms by fœtor of the sputa. There appear to be two classes of cases that present this phenomenon. In one the expectoration is extremely offensive like the putrescent odor of pulmonary gangrene, but neither clinically nor *post mortem* can be discovered any other changes than those of chronic bronchitis. Such a case was described by Andral, and similar observations have been reported by others. In this class of cases the fœtor of the sputa may be transient or persistent.

In the second group of cases there are other changes of a serious nature which appear to act as predisposing causes. These are bronchiectasis, gangrene of the lung or pulmonary cavities. Dittrich, who first described putrid bronchitis minutely, held the view that it resulted from stagnation of the secretions within bronchiectatic or true pulmonary cavities. Traube's cases confirmed this view in the main, since in 10 out of the 12 cases gross changes, e. g., gangrene, bronchial dilatations or vomicæ, were discovered. In the two others nothing more than chronic bronchial catarrh was demonstrated.

The feature common to all cases is the intensely stinking odor of the sputa and breath. It closely resembles that of pulmonary gangrene and is so intense as sometimes to fill the sick-room. It is attributed to decomposition of the albuminous constituents of the secretions *in situ* occasioned by the action of putrefactive organisms. The *sputa* are usually abundant and when allowed to stand, separate into three layers. The uppermost consists of froth, mucus and pus, the middle of a turbid, brownish serum, and the bottom of a thick, dirty-looking sediment.

This bottom layer contains the so-called *Dittrich's plugs* which emit an intolerably stinking odor. These are granular masses of a yellowish or yellowish-brown color running in size from that of a millet seed to that of a small bean and composed of cellular *débris,* fat crystals and bacteria. The sediment furthermore contains epithelium, pus and red blood cells, fat drops, crystals of the fatty acids, leucin, tyrosin, Charcot's crystals and various organisms of putrefaction, and it therefore closely resembles the bottom layer of gangrenous sputum. It does not contain hæmatoidin crystals unless pulmonary gangrene is present.

Decomposition of the bronchial secretions *in situ* is essential to the production of fœtid bronchitis but is hardly likely to take place without some morbid anatomical change favoring retention of the sputa. Accordingly there is generally found *post mortem* either gangrene of the lung or a bronchiectatic or tuberculous cavity. When bronchial dilatation is discovered it is not always clear whether this was the antecedent condition or developed in consequence of the weakening effect of the putrefactive process upon the bronchial wall.

It would seem, therefore, that the term putrid or fœtid bronchitis ought to be limited to cases which at the autopsy display no other changes than those of chronic bronchitis, and which during life show no other findings than those of chronic cough with decomposed and stinking expectoration. Inasmuch, however, as it is not always possible to determine clinically whether bronchial dilatation or even more serious structural lesions are associated, it has become customary to include among putrid bronchitis all cases which disclose Dittrich's plugs in the sputum.

This form of chronic bronchitis is usually encountered either as a terminal event or as a transient condition. The invalid who has become greatly reduced by his long-standing disease is incapable of resisting infection either through the accidental access to the tubes of saprophytic organisms or in consequence of contact with other bronchitics having a fœtid expectoration. Fraenkel states that he has repeatedly seen one patient after another become infected in this manner.

One of Traube's patients had suffered from chronic bronchitis for sixteen years since the age of twelve and twice had experienced transient fœtor of the sputum. His condition upon entering the hospital was one of great prostration and it was noted that his sputa were very offensive. Physical examination disclosed the signs of chronic bronchitis, but the symptoms were those of profound septicæmia: fever, dyspnœa, sweating and intense weakness. His condition grew steadily worse and on the third day after his admission he died. The *post-mortem* examination revealed, "putrid bronchitis, no cavities, no gangrene, recent pneumonia and pleurisy, diphtheritic inflammation of the smaller bronchi."

The following case which may be regarded as one of putrid bronchitis was seen by me at the Cook County Infirmary in December, 1902. The man, a foreigner, aged thirty-nine, had worked as a stoker up to May, 1902. As nearly as could be gathered from his indefinite account of his illness he then became ill with cough and fever which laid him up in bed for a few days or a week. He then tried to resume his work but was not able to attend to his duties for more than a day or two at a time.

After being thus ill for a few weeks he was admitted to the Cook County Hospital for Consumptives where he was examined by the resident physician, Dr. MacHugh. This physician stated that on admission the expectoration was horribly fœtid, composed largely of pus and contained numerous mixed organisms but no tubercle bacilli. The chest examination showed nothing more than the ordinary signs of bronchitis.

Accordingly the man was transferred to the Infirmary where he had remained up to the date of my examination. During all the intervening months the sputa were so stinking that other patients objected strongly to sleeping in the same apartment and the man held a handkerchief in front of his mouth whenever compelled to cough. His expectoration was abundant, and troubled him chiefly at night soon after going to bed and upon waking in the morning. He had lost about forty pounds in weight.

Upon approaching the man for the purpose of examination I at once perceived an indescribable odor somewhat fæcal in character, emitted from his mouth, although it was stated that the fœtor was not so intense as some months previously. The man was of more than average height and of large frame, but much emaciated, and stood with his shoulders drooped forward, his back bent and his prominent shoulder blades spread wide apart.

The chest was large and bony, but so sunken at the apices that the clavicles showed prominently (Fig. 1). In marked contrast to the capacious thorax was the hollow, emaciated abdomen. When told to breathe deeply the man raised rather than expanded his chest. The percussion-note was resonant excepting in the right interscapular region and at the left apex behind, where there was slight but appreciable impairment. During percussion it was distinctly perceived that the walls of the thorax had lost their natural elasticity.

Upon auscultation the breath-sounds were found exaggerated and rough, but nowhere bronchial, and there were no râles. The voice-sounds were bronchophonic and in this respect corresponded with a distinct increase in vocal fremitus.

From the history, symptoms and physical signs the case seemed to be one of fœtid bronchitis, though whether or not dependent upon bronchiectasis could not be determined. Dr. MacHugh, the Medical Superintendent of the Consumptive Hospital who had examined the man at the time of his admission, was certain that the resonance of the lungs had become appreciably diminished in the intervening months; and hence it seemed not unlikely that more or less fibrosis of the lungs with consequent dilatation of the bronchi had taken place.

The expectoration was subsequently collected for me and examined at the Columbus Medical Laboratory. The following is the report. Amount, 465 cubic centimeters, semi-fluid, grayish color, stinking odor and separates into two layers the upper being one-third the thickness of the lower. The upper one is brown with a greenish fluorescence, while the lower is thicker and grayish in appearance. The upper is fluid, but shows the stringy consistence of mucus when poured. The lower thick layer is quite granular and contains numerous masses which are quite yellow or yellow gray in color and from 1 to 3 centimeters in size. These are the plugs of Dittrich.

Microscopic Examination. — Unstained, numerous partly disintegrated cells, pus and epithelium. Bacteria are also seen in great numbers, bacilli predominating, many of which are motile. Stained specimens (methyl blue) show mixed cocci and bacilli. Among the cocci, staphylococci and a few sarcinæ are seen. A thin long bacillus predominates. Gram's stain shows cocci, and a few of the bacilli hold the stain. Iodine stain shows a moderate number of larger bacilli which give a bluish color with this stain.

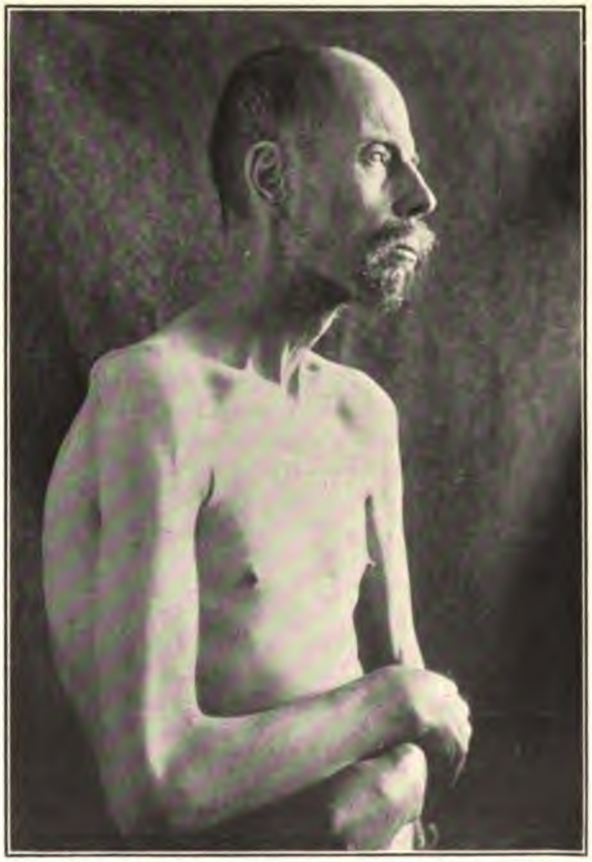


FIG. 1.—Photograph of patient suffering from putrid bronchitis.

Examination of the plugs shows that they are composed of masses of leptothrix-like organisms matted together with granular and fatty detritus. Only a few pus cells are found in these masses. The iodine stain gives these a bluish color and in addition they are quite granular. Staining for bacillus tuberculosis is negative and pneumococci cannot be distinguished neither can streptococci.

Cultures.—From the mixed sputum a proteus form is isolated from the colonies that are most numerous on agar. The cultures show a thin long bacillus which grows rapidly, is smeary, and liquefies gelatine. There is a marked odor of putrescence to these cultures. Staphylococcus albus was also isolated. From the plugs the bacillus proteus was isolated and it does not seem possible to determine if this is the leptothrix-like organism in the plugs or if that organism has failed to grow in the media.

Physical Signs. Inspection.—In the early stage of chronic bronchitis there is nothing in the patient's appearance worthy of note. Even after the disease has lasted for years and the general health has been undermined, inspection may fail to detect more than a certain degree of emaciation and a look of ill health. The respirations may appear difficult, the chest may assume the barrel-shape of emphysema and there may be epigastric pulsation, visible cutaneous capillaries and other signs of circulatory embarrassment. In some instances the supraclavicular and infraclavicular regions look abnormally sunken and at first glance the chest may resemble that of pulmonary phthisis, but closer inspection generally discovers greater rotundity and less flattening than is seen in consumptives.

Palpation is for the most part of negative value. Pectoral fremitus may be unchanged, or, if emphysema is also present, is likely to be diminished. Occasionally when the tubes are filled with secretions the hand laid upon the base of one or both lungs detects a vibration during inspiration known as rhonchal fremitus and due to vibrations communicated to the mucus by the passage of air. The nature of such fremitus may generally be determined by having the patient cough, since, if it is generated in the air-tubes, it is likely to disappear after the act of coughing has dislodged the secretions.

Percussion.—Chronic bronchitis, like the acute, is a disease which does not lead to solidification of lung-tissue and therefore percussion is of value in determining the retention of pulmonary resonance. The note is clear and when emphysema also exists is hyperresonant. Chronic bronchitis of the capillary tubes may lead to their occlusion or even to areas of atelectasis, and when such is the case at one or the other base, the percussion note lacks full resonance or may be actually dull in a circumscribed area. The act of coughing is likely to clear up such impaired regions when the percussion note becomes resonant.

Auscultation is of chief value and often furnishes the only reliable information. The breath-sounds may be vesicular, but in most cases when not obscured by adventitious sounds are rougher than normal, while the expiratory murmur is apt to be prolonged.

In dry bronchitis there may be no râles, but in the majority of cases of chronic bronchial catarrh auscultation usually discovers râles. These may be dry or moist or made up of a blending of both kinds. Although they may be audible in any portion of the chest, they are generally most abundant and constant at the base, especially behind.

Dry râles or rhonchi are variable in pitch and loudness according to the caliber of the tubes in which they are generated. Sonorous and large bubbling râles may be heard over the large bronchi when secretions are fluid and abundant, but in most cases of chronic bronchitis the chest emits whistling and squeaking rhonchi which conceal the respiratory murmur and are often interspersed with subcrepitant râles. These râles may be heard with either or both inspiration and expiration and vary in distinctness and quality according to the depth of respiration. Occasionally an unusually deep inhalation is accompanied by a squeak or loud click that gives the impression of a small tube having been suddenly and forcibly opened to the entrance of air.

In some instances the chest is filled with such a multitude and variety of sounds of differing pitch and intensity as to be fairly bewildering and a source of wonderment to the student. Finally when the râles consist chiefly or wholly of moist fine or medium-sized bubbling, their true nature as bronchitic may be determined by having the patient give a few energetic coughs when they are found to have diminished in number or disappeared.

Diagnosis.—This is not a matter of great difficulty in cases displaying the physical signs described above, especially when rhonchi are present at the base of the chest. In dry bronchitis, or in the chronic Winter cough without râles, the diagnosis rests upon a history of chronic cough, upon the ætiological data of age, occupation, repeated attacks of acute bronchial catarrh, etc. and the retention of pulmonary resonance. This last is a matter of the utmost importance in all cases of chronic bronchitis whether râles are or are not present. In occasional instances rhonchi are few or wanting and the lungs give forth moist or dry râles of a crackling character, the former resembling those of pulmonary phthisis and the latter suggesting pleuritic adhesions.

Consequently the following points are to be borne in mind. (1) The râles of bronchitis are usually bilateral, though they may be more numerous and pronounced on one side than the other. (2) They tend to be more distinct and abundant as auscultation is made from above downward. (3) They are usually more or less widely distributed throughout both bases. (4) They are not likely to be associated with loss of pulmonary resonance, unless the bronchitis has led to atelectasis. When such is the case it is generally found that the dull area clears up after cough or a few forcible inspirations.

Differential Diagnosis.—(*A*) Pulmonary tuberculosis is the disease with which chronic bronchitis is most likely to be confounded, and hence the following are the main differential points.

(1) *Age.*—Tuberculosis is more common in early life, i. e., between the fifteenth and thirtieth years, chronic bronchitis more frequent in middle and advanced age.

(2) Tuberculosis occasions a more marked and rapid cachexia.

(3) Tuberculosis is usually attended by fever while in chronic bronchitis pyrexia is generally absent.

(4) Pulmonary phthisis is unilateral or when double is more advanced on one side, and hence the signs of disease are confined to one lung or predominate there.

(5) The affected apex is sunken and more or less immobile during respiration.

(6) One or both apices are dull or, if resonant, are tympanitic owing to the presence of a cavity.

(7) The breath-sounds are bronchovesicular, bronchial or cavernous according to the degree of the pathological changes.

(8) If râles are audible they are moist bubbling, or of a subcrepitant character, and are usually most numerous at or near the apex rather than at the base.

(9) If doubt still exists, recourse may be had to examination of the sputa which, in tuberculosis, are likely to contain the characteristic bacilli or possibly also fragments of lung tissue.

(*B*) Cases are sometimes encountered in which old pleuritic adhesions at the base of one lung or more rarely at the bottom of both lungs occasion some difficulty in diagnosis. Error may usually be avoided by due attention to the patient's history, symptoms and the following points:—

(1) Pleuritic friction râles are likely to be circumscribed.

(2) They are apt to be heard during both respiratory acts and when not creaking have a fine dry crackling quality.

(3) They are not affected by cough, i. e. do not diminish or disappear.

(*C*) Pulmonary œdema may simulate chronic bronchitis and the acute form may be mistaken for bronchorrhœa serosa.

(1) In pulmonary œdema there are likely to be the history and symptoms of cardiac or renal disease rather than of chronic cough and expectoration.

(2) The chest is filled with fine moist bubbling râles and the patient coughs up frothy, perhaps blood-tinged sputum.

(3) In the acute form the onset is usually sudden and may follow unwonted physical effort.

(4) Chemical examination of the expectoration shows that it contains albumin instead of mucus.

(*D*) Bronchial or nervous asthma may usually be distinguished from asthmatic bronchitis by the history and especially by the circumstance of entire freedom from cough and expectoration during intervals between the asthmatic paroxysms.

Prognosis.—Chronic bronchitis is a serious affection in its ultimate rather than in its immediate effects. The disease possesses no inherent tendency to recovery and if it is not checked by treatment it will inevitably progress to the ultimate disablement of the patient and in time to death either through the secondary changes induced in lungs and heart or through some of the complications to which it predisposes.

Even in the early stage of the complaint the prospect of complete recovery is not always encouraging and depends upon the removal of all conditions that favor its continuance which is manifestly not possible in every instance. If the sufferer is able to pass the inclement months in a suitable climate he may escape his recurring winter cough or suffer but slight inconvenience therefrom. When such a change of residence is not possible there is small prospect of a cure by the most persistent medicinal treatment.

The development of emphysema and asthma especially in persons of middle or advanced age generally renders futile all attempts at a cure. In the

young, on the contrary, these complications may sometimes become
with increasing age (Fowler). The existence of heart or renal d
further lessens the likelihood of an arrest of chronic bronchitis or
material betterment by the therapeutic measures at our command.

When at length the patient has become cachectic he is liable
either in consequence of exhaustion or of an acute exacerbation of
chitis. He is also exposed to the danger of an intercurrent pneumo
ertheless, the disease is essentially chronic and, barring some i
affection, it is likely to persist for many years.

CHAPTER IV

PLASTIC BRONCHITIS

Of other terms applied to this disease the most common are fibrinous and croupous bronchitis. By Biermer it was called bronchial croup and by some authors it is termed pseudo-membranous bronchitis. I have chosen the appellation plastic because of the uncertainty of the casts being always composed of fibrin.

The affection is characterized by the expectoration of casts of the bronchial tubes and in the primary or essential form is exceedingly rare. So rare, indeed, that as Lenhartz remarks: if the importance of the disease were to be estimated by the frequency of its occurrence it might be said to possess very little practical value.

The rarity of this affection may be judged of from the statement made by the same author that up to 1878 Biermer was able to collect from the literature only 58 cases, while up to 1874 Lebert had only succeeded in collecting 17 acute and 27 chronic instances. There is a discrepancy between these figures and according to Riegel the explanation must lie in the fact that no distinction was made by Biermer between primary and secondary cases.

The most common classification of plastic bronchitis is into acute and chronic. The former term is applied to cases in which but a single attack occurs, while chronic, is employed to designate cases in which the expectoration of bronchial casts takes place repeatedly through a period of weeks or months. There can be no objection to the term chronic if the characteristic symptom is experienced at short intervals during an extended period. If, on the other hand, it is applied to cases showing this symptom at intervals of years or months, then it is objectionable because misleading.

It is deemed preferable, therefore, to divide plastic bronchitis into primary and secondary, since such a classification is in accordance with our present, though rather incomplete, knowledge of its ætiology. By *primary* or essential cases, are meant all those that arise independently of any other affection, while in *secondary* are included cases that arise in the course of or seem to depend on some antecedent affection.

Bettmann's classification of his collected cases, is as follows:

I. Chronic bronchitis with expectoration of branching casts of the bronchial tree: 27 cases.

II. Acute bronchitis with expectoration of branching casts of the bronchial tree: 15 cases.

III. Cases in which branching casts were not expectorated, but in the bronchi at autopsy: 6 cases.

IV. Cases in which the casts expectorated showed no dichotomo ing: 11 cases.

V. Expectoration of branching casts in organic heart disease:

VI. Expectoration of branching casts in pulmonary tuberculosis

VII. Expectoration of small casts, often nonbranching, in associ asthma: 5 cases.

VIII. Formation of casts in the bronchi in association with œdema following thoracentesis: 4 cases.

IX. Cases whose classification is doubtful because of incomplet 6 cases.

Ætiology.—As already stated we have to distinguish a prim secondary plastic bronchitis. The causation of the former is stil of uncertainty, although the association of the secondary form wi infectious diseases suggests that in the primary, or as it is calle variety, micro-organisms may also be the determining cause.

The secondary form has been encountered in diphtheria, lobar p measles (Jäger), scarlatina (Anderson, Möller), variola, typh (Eisenlohr, Strümpell), articular rheumatism, tuberculosis and It is not difficult to understand its occurrence in pneumonia whe sider that the inflammatory exudate caused by Fraenkel's pneum peculiarly rich in fibrin; neither is it strange that a bronchitis o coccus origin unassociated with pneumonia should occasionally be ized by the formation of bronchial casts.

The Loeffler bacillus also occasions a croupous exudate or false and Lenhartz suggests that some of the primary cases may have been of masked diphtheria. Of 220 fatal cases of diphtheria examined cilman, Mallory and Pearce fibrinous bronchial casts were prese The determination of such an ætiology is, as remarked by Hoffma utmost importance in its bearing upon prognosis and treatmen länder's pneumococcus was identified in one case of fibrinous (Magniaux) both by cultures and in the microscopic examination o

Hoffmann cites the cases reported by Picchini which occurre laborers who had worked in a sewer and in which he identified thre bacteria one of which when injected into rabbits produced a ha inflammation of the trachea. Consequently, although other organ those mentioned have not been definitely recognized, we must as still other organisms are capable of producing fibrinous bronchitis.

Its occurrence in chronic catarrhal bronchitis, as well as in t and heart disease is as yet not easy to explain excepting on the of some bacterial influence. The disease has been known to follow lation of irritating fumes of ammonia (Lenhartz) and of hot smo berger) while Beschorner has narrated the highly interesting case cian who developed an attack of fibrinous bronchitis apparently as of becoming suddenly chilled. This patient when greatly overhea the cooling room at the Berlin Exposition and there remained ab minutes. He soon thereafter perceived a sensation of tickling and burning beneath the sternum and inclination to cough, and two days later suffered from the distressing symptoms of an acute plastic bronchitis. As remarked by Lenhartz in commenting upon this case we are able to ascertain the origin of such cases but we can only conjecture concerning the rôle played by infection. Patten and Herzog have reported a case in which they failed to obtain by culture anything but "colonies which consisted of large round micro-organisms, probably yeast cells or a species of torula."

Morbid Anatomy.—There can scarcely be said to be any morbid anatomical appearances distinctive of plastic bronchitis. Such changes as are found belong to the disease to which the expulsion of casts is secondary. Fraenkel lays great stress on the discovery of circumscribed losses of epithelium protecting the surface of the bronchial mucosa, as has been noted by some investigators (Kretschy, Weigert). In consequence of this desquamation of the epithelium there occurs an exudation of lymph which then undergoes coagulation and spreads over a considerable surface of the mucosa. This is the counterpart of what takes place, therefore, in laryngotracheal diphtheria. Fraenkel holds that such a loss of epithelium is essential to the production of fibrinous molds and should receive special attention in future autopsies.

There have not been many necropsies in cases of plastic bronchitis, but in such as have been made, the lungs have shown no changes that threw light on the causation of the disease in question. The air-tubes in some part of the lungs (in the middle and right lower lobes in Kretschy's case) are found to contain fibrinous masses which may partially or completely block the bronchioles and are here and there detached from the mucous membrane. These masses are covered by mucus and by a frothy or bloody secretion. The mucosa underneath is bare of its epithelial covering. The lobules supplied by the bronchi are either collapsed or emphysematous, depending upon the completeness with which the latter are blocked.

The Casts.—These molds or plugs may be either hollow or solid cylinders which vary in length and thickness. In length they may vary from 2.5 cm. to 20 cm. (1 to 8 inches) and their diameter depends upon the tube from which they come. Their outer surface is generally covered with more or less mucus and frothy blood, and when expectorated they are usually rolled up into balls or roundish masses. Upon their being manipulated in water they unroll and assume a form resembling the branch of a tree (Figs. 2 and 3). The stem of the cast is thicker than its subdivisions and has been known to measure 1.5 cm. ($\frac{3}{5}$ of an inch) in diameter. The branchings or subdivisions of the stem taper down to a small size corresponding to the caliber of the bronchioles into which the casts extended. The free extremities of the terminal twigs may show slight bulbous enlargements indicating their formation within the infundibula.

Examined microscopically the casts are seen to be made up of layers placed one upon another, as many as eight such laminæ having been observed. Held within the meshes of the fibrin generally composing the casts, are leucocytes, red blood-cells, bacteria and epithelium which may be well preserved or partly degenerated. Charcot's crystals may sometimes be seen and Flint reported the detection of hæmatoidin crystals.

The material composing the casts is generally stated to be fibrin as proven by chemical analysis and Weigert's fibrin stain. On the other hand cases have been reported (Neelsen, Grandy) in which the molds were undoubtedly composed of mucin. Accordingly it seems justifiable from this standpoint to divide plastic bronchitis into two groups, namely, *bronchitis fibrinosa* and *bronchitis pseudo-membranacea mucinosa,* as is done by Fraenkel.

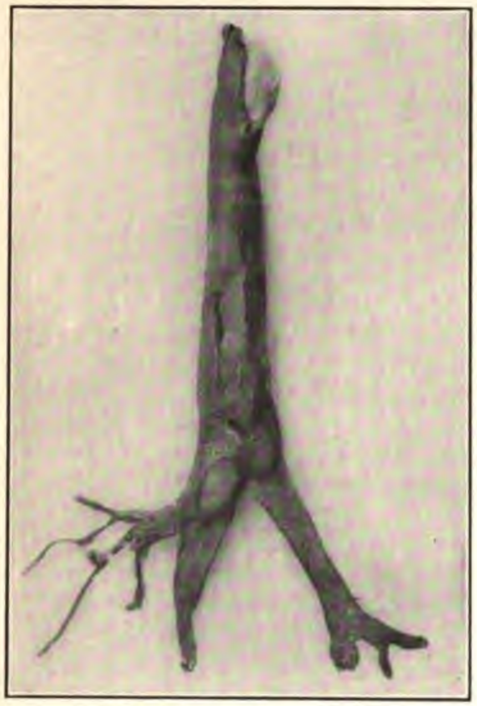


FIG. 2.—Bronchial cast.

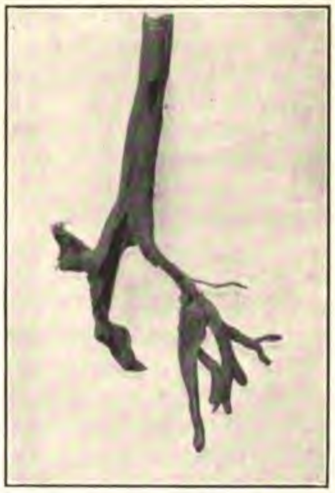


FIG. 3.—Bronchial cast.

Inasmuch as these two groups may have a different ætiology it is necessary to determine in every case to which group it belongs. Hence must be ascertained the nature of the material composing the casts.

Fibrin is oxyphile and stains with acid dyes. Mucin is basophile and stains with basic dyes. Weigert's stain for fibrin, sometimes called the Weigert-Gram stain, is essentially the ordinary procedure of Gram's stain with the addition of an anilin oil decolorization. The method is as follows: (1) Stain with anilin water-gentian violet. (2) Gram's iodin solution. (3) Further decolorization in anilin oil 2 parts, xylol 1 part. (4) Mount in xylol or xylol balsam.

This imparts a blue color to fibrin, whereas mucin becomes gray. Moreover, fibrin is digested in artificial gastric juice while mucin is not. It would certainly seem, therefore, that the diverse results obtained by competent observers cannot be due to error but indicate that bronchial casts may be made up either of fibrin or mucin. Finally, Liebermeister has recently reported an analysis of the recorded cases of plastic bronchitis. He found fibrin was present in all, while in 7 there was also mucin.

Symptoms.—It should be remembered that some authors distinguish both an acute and a chronic form of this disease, classifying under the acute those cases in which there is but a single sudden attack, and under the chronic those characterized by a recurrence of attacks extending over a period of weeks, months or years. In both forms the features of the paroxysms are essentially the same and therefore the following description applies to either the acute or the recurrent form in the sense employed by German writers.

The striking symptoms of an attack are dyspnœa, cough and the expectoration of a false membrane. The difficulty of breathing is similar to that of an asthmatic paroxysm, but is even more intense, being as a matter of fact so urgent that the patient appears in imminent danger of suffocation. The sufferer makes violent and ineffectual attempts at inspiration, grows oftentimes greatly cyanotic and perspires profusely, while his distress is still further intensified by fits of prolonged and violent dry coughing which may be repeated for hours or days before expectoration results in relief.

Such an attack may set in abruptly or it may be preceded by symptoms of an acute bronchitis, as substernal burning, irritation and oppression. In primary cases there may be a single chill or repeated chills, and fever, a temperature of 104° F. having been more than once reported (Patton and Herzog, e. g.). Not only is the respiration extremely difficult, but its rate becomes accelerated, even as high as 74 respirations per minute having been noted. The pulse-rate is also increased to 120, 140 and even 160, and in strength the pulse is diminished.

If at this time the patient's chest is examined there may or may not be recognizable signs of the nature of the difficulty. If a main bronchus is occluded, the corresponding half of the thorax is immovable during respiration, the percussion note is resonant and breath-sounds are abolished. If, as more frequently happens, but a limited area is affected, this portion of the lung may yield signs of atelectasis, i. e., dullness and absence of respiratory murmur, or it may be emphysematous and disclose very equivocal physical signs. Whistling râles over a circumscribed area have been heard and in some cases there has been a peculiar rattling or flapping sound thought to be caused by the movements of the fibrinous membrane.

After a variable length of time, in fortunate cases twelve hours from the onset (Kretschy), or in some instances not until after the lapse of several days, the violent and protracted fits of coughing result in the expulsion of the cast. As already stated, this looks like a ball or mass which is only recognized as a mold of the air-tubes after having been gently unrolled in water. The extrusion of the offending material may be preceded or attended by hæmoptysis sometimes of considerable amount.

With the expectoration of the cast the patient's condition undergoes a striking change for the better. His dyspnœa and sense of imminent asphyxia cease, his cough subsides and he falls back exhausted but relieved. Symptoms of circulatory embarrassment gradually vanish and after a refreshing slumber the patient either regains his former health or enjoys only a respite

of uncertain length from a repetition of his sufferings. In som
the period of freedom lasts but a few days, the casts then refo
patient is plunged into another paroxysm of threatening suffocat
to the first.

No rule can be stated regarding the number and frequency of s
Walshe recorded a case which, with intermissions, extended over
fourteen years, and Peacock one of a boy of eleven who experienc
ease four times during five years, each recurrence persisting for
(Wilson Fox). West relates the case of a woman whose paroxys
with such regularity at 2 o'clock each night as to suggest asthma
other of his patients, an emaciated girl of nine years, experienced
during the month she was under observation, sometimes once in
sometimes as often as several times daily.

It is apparent, therefore, that the attacks may return with su
quency and intensity to wear out the patient, or that only one a
may be experienced. Individuals who manifest these distressing
at intervals over a period of years may either be quite well dur
terim or may suffer more or less from chronic bronchitis of t
catarrhal type. In the recurrent or chronic form moreover there is
of fever.

The case reported by Patton and Herzog was an instance o
variety and is sufficiently characteristic to warrant its insertion he
bottler, aged forty, was well until two years before he came und
tion when, by working in a wet place, he took cold. During thes
he had repeated attacks of bronchitis which lasted a week or ten
and were characterized by severe rasping cough. Expectoration wa
tively free and there was no pain in the chest. About ten weeks
last attack he began to cough and to expectorate large masse
tenacious sputum which was at times accompanied by some blo
four weeks before the expulsion of the bronchial casts he deve
fever, great dyspnœa and a distressing cough. Examination sho
ished expansion and some dullness over the lower two thirds of
compared with the right lung. This attack persisted between thr
weeks, in fact up to the time of the expectoration of the casts.
of his cough, dyspnœa and fever, which at times exceeded 102°
him to his bed. Three days before the morning on which the ca
pelled he spat up considerable blood, and on this eventful mo
cough and difficulty of breathing increased, his temperature rose to
at length, after a specially violent effort, he expectorated a large
proved to be a bronchial cast mixed with mucus and quite a little

Within a short time thereafter, examination of his chest by hi
physician showed that air was entering the base of the left lung
parative freedom, while the percussion note was of nearly norma
and mucous râles were present over both lungs. The patient's r
rapid and he soon passed from further observation.

Physical Signs.—Inspection may in some instances detect r
abolished respiratory movements over one side of the chest or

circumscribed portion of one side. Such discovery together with other objective and subjective symptoms is very suggestive.

Palpation affords no positive information, but if tactile fremitus is absent over a limited area this may greatly strengthen other signs.

Percussion.—The chest may be resonant the same as in ordinary cases of chronic bronchitis or there may be dullness limited to an area in which collapse has taken place. It is very possible, however, for such atelectatic area to be too deeply situated for recognition. In some instances, on the other hand, percussion may disclose circumscribed tympany due to localized emphysema, but the recognition of such an area in the midst of surrounding pulmonary resonance of normal quality is not an easy matter.

Auscultation.—An absence of respiratory murmur over a whole lung which at the same time is resonant, or over a circumscribed area of one lung, which area may be either dull or tympanitic, is very suggestive and when associated with inspiratory dyspnœa and other significant symptoms may enable one to make an inferential diagnosis. There may also be râles of chronic bronchitis.

Diagnosis.—It goes without saying that the only positive means of diagnosing this affection lies in the detection of expectorated casts. In most of the cases, in which but a single attack occurred, the diagnosis was not made prior to the expulsion of such casts. In recurring cases the history of previous attacks may aid in the recognition of the true nature of the paroxysm. Theoretically, there should be some of the physical signs described of sufficient definiteness to lead to a diagnosis, but in practice the most skillful clinician may make repeated examinations without being able to do more than surmise the existence of this formidable malady. Should the symptoms described occur in the course of diphtheria or acute fibrinous pneumonia or bronchitis, the fact of the association of these affections would render the existence of plastic bronchitis probable.

Differential Diagnosis.—Not all authors consider the differentiation of plastic bronchitis from other affections with which it may be confounded, probably because they consider the nature of the case assured by the expulsion of the cast. Yet, prior to the occurrence of this only pathognomonic sign, it is quite possible for the attack to be mistaken for any one of the following conditions: (1) A paroxysm of bronchial asthma. (2) Stenosis of the larynx or trachea. (3) Pneumothorax. (4) Compression of a bronchus by tumor or aneurysm. (5) Acute pneumonia.

(1) (*a*) In asthma, there is a history of previous attacks which subsided without the expulsion of casts. (*b*) In asthma the dyspnœa is chiefly expiratory, while in plastic bronchitis it is inspiratory. (*c*) In asthma the chest is distended in the position of permanent inspiration. (*d*) In asthma there is hyperresonance, while in fibrinous bronchitis there may be dullness or tympany over a circumscribed area. (*e*) In asthma the lungs are filled with whistling râles, while, although râles may be present also in plastic bronchitis, there is a diminution of respiratory murmur over one lung or a limited portion of one lung.

(2) (*a*) In laryngeal or tracheal stenosis the dyspnœa is also inspiratory but the neck is extended with the head thrown backward with alternate lifting

and falling of the larynx. (*b*) There is likely to be inspiratory s
laryngoscopic inspection may reveal the cause of the obstruction.
laryngeal obstruction is due to a diphtheritic exudate, inspection i
discover a similar false membrane in the fauces. (*d*) Stethoscopi
tion of the trachea may, when the stenosis is here, detect the s
obstruction. (*e*) Although in stenosis there may be stertorous bre
chest is not filled with râles, as may be the case in fibrinous bron
any diminution of respiratory murmur is general over both lungs r
unilateral or circumscribed.

(3) Pneumothorax is differentiated by the following points.
probable history of previous tuberculosis which although it has been
with plastic bronchitis, is very rarely so associated. (*b*) Pne
usually sets in abruptly with sharp pain in the affected side and th
develops somewhat later. (*c*) There is hyperresonance, exceptio
ness, over one half of the thorax which is likely to be distended a
both by inspection and mensuration. (*d*) The heart is displaced t
site side.

(4) In cases in which a bronchus is compressed by aneurys
tumor the real nature of the case ought to be disclosed by (*a*) the
(*b*) careful physical exploration of the chest. (*c*) If doubt still
X-ray will probably clear up the diagnosis.

(5) Acute pneumonia is also mentioned as one of the diseases f
plastic bronchitis is to be distinguished, and the case reported by
Herzog shows the closeness of the resemblance in some respects. I
there was high fever, together with dullness and enfeebled breath-s
the lower two thirds of the left lung. One should also remember
monia and fibrinous bronchitis may be associated. For the diagnos
pneumonia the reader is referred to that chapter.

It may here be stated that aside from the less pronounced co
symptoms in acute plastic bronchitis the chief differences are to b
signs within the lungs. In pneumonia there are (*a*) dullness, (*b*
breathing, (*c*) the characteristic crepitant râle, (*d*) perverted pu
tion ratio. (*e*) In fibrinous bronchitis there may or may not be cir
dullness, but if the note in a given area is dull it is likely to be asso
diminished or absent rather than bronchial respiration.

Finally, the observer should be on his guard not to confoun
casts of the kind here considered with blood-clots that have been m
casts within the air-tubes. (*a*) Blood-coagula are not stratified.
fibrinous molds are only bloody upon their external surface, whil
found on microscopic examination to have red and white blood-cells
throughout their substance. (*c*) Although Fraentzel's case, that
sumptive soldier who expectorated true fibrinous casts, proves t
association of tuberculosis and plastic bronchitis, still such a com
rare, whereas hæmoptysis and pulmonary tuberculosis are freque
itants. This is not be construed as meaning that hæmoptysis
bronchitis may not be associated, but that the concurrence of the f
signs of tuberculosis would, in the absence of microscopic exami

one in determining the nature of the molds when these are coagulated blood.

Prognosis.—Patients rarely if ever die from direct result of the casts, i. e., from asphyxia, but from the primary disease. Thus in Magniaux's case that of an eleven-year-old boy death was probably due to the diphtheria although a large cast extended from the trachea into the bronchi. In a nine-year-old girl observed by West the necropsy disclosed miliary tuberculosis of the lungs with chronic bronchitis and old pleural adhesions, so that the fatal termination was ascribable not to the formation and expulsion of bronchial molds although they necessarily occasioned great exhaustion. Nevertheless, an attack of plastic bronchitis cannot be declared devoid of danger either from the exhaustion attending the violent expulsive efforts or from suffocation in consequence of retention of the casts.

Unfortunately the extent and size of the molds cannot always be determined *intra vitam* and hence the physician should always give a guarded prognosis. The liability to recurrence is also uncertain and renders the outlook contingent thereon. If previous attacks have been experienced or if a chronic disease seems responsible for the formation of the casts, then recurrences may be expected, but how soon or how many cannot be even surmised.

CHAPTER V

TREATMENT OF THE VARIOUS FORMS OF BRONCHITIS

Prophylaxis.—This includes the avoidance, as far as possible, of all conditions predisposing to bronchitis. In addition it embraces all measures calculated to fortify individual resistance against the injurious influences that have been discussed under the subject of ætiology. Young persons of a delicate constitution who display especial vulnerability of the air-passages should be subjected to all measures likely to harden their tissues and lessen their liability to colds. They should accustom themselves to a cold morning bath or shower which diminishes the sensitiveness of the skin to sudden changes of temperature. For the same reason they should avoid the wearing of too heavy clothing, the muffling of the neck in furs, tippets, etc., since, by causing perspiration and relaxation of the skin, they predispose to a cold upon exposure to raw winds. In particular, they should be warned against wetting of the feet, sitting in draughts, passing from a heated ballroom to the outside air without a wrap and the many other imprudences of which the young are guilty by reason of vanity or thoughtlessness.

Disorders of the nose and throat should receive particular attention, since many a child owes its frequent repetition of colds and coughs to the presence of adenoids or enlarged tonsils. It is necessary also that the excretions be kept active and the child not allowed to indulge often in candy and rich or indigestible articles of food. In my opinion there is an intimate association between catarrhal disorders of the alimentary tract and those of the respiratory mucous membranes, and hence whatever predisposes to the former tends also to the production of the latter.

Old people and invalids are very apt to overheat their apartments and to exclude fresh air under the impression that by so doing they lessen their liability to colds. As a matter of fact the impurity of the atmosphere thus occasioned predisposes their air-passages to the harmful action of germs as well as lowers their tissue-resistance. Consequently, whenever they pass from their own ill-ventilated, overheated apartments into those of lower temperature they are very apt to become chilled and to develop a cold. For the same reason it is a mistake to permit a child's nursery to become too hot and close. Fresh cool air is a safeguard against colds and persons who accustom themselves to living in such an atmosphere do much to protect themselves against bronchitis.

As long as the days are warm and pleasant windows and doors should be left freely open for the admission of sunlight and pure air. When cold weather sets in and artificial heat becomes necessary, the house or office should be thoroughly ventilated several times a day. Persons who, on account of age or infirmity, must remain housed during inclement weather should take advantage of every fine day to get out of doors lest their general health suffer and their liability to bronchitis be increased.

Individuals recently recovered from the *grippe* or other acute infection should not be exposed to raw, chilling winds until the sensitiveness of their mucous membranes has disappeared. Whenever practicable they should insure their complete convalescence by a temporary sojourn in a mild climate. This suggestion is especially applicable to children who have but lately passed through an attack of measles or pertussis. Men who spend their leisure hours in public resorts and whose air-passages begin to show the injurious effects of alcohol and tobacco smoke should be advised of the risk they run and urged to change their mode of life.

Proper clothing is a matter of great importance both as regards prevention of colds and the protection of chronic bronchitics. Persons obliged to work in steam-heated offices or elsewhere should not wear too heavy underclothing lest they keep in a perspiration, and when they pass out doors they should be careful to protect themselves against sudden chilling of the body. Children, in particular, are often too warmly clad in the erroneous notion that by so doing they are protected against draughts. In reality they perspire freely and increase their liability to catarrhal disorders. Sensitive old people on the other hand must guard against chilliness when in the house by throwing wraps about their shoulders. Finally, all individuals with sensitive air-passages should take care not to get wet feet but should subordinate their vanity to prudence in all such matters.

Abortive Treatment of Acute Bronchitis. — The bronchitis symptomatic of measles and other specific infections cannot be aborted, but may be mitigated in its severity by due attention to certain measures. A simple or nonspecific tracheo-bronchial catarrh may on the other hand be sometimes thrown off or cut short by a prompt resort to antiphlogistic and eliminating remedies.

A robust adult may appreciably relieve the tightness of the chest and cough by the application of a mustard plaster or turpentine stupe to his chest and by a dose of calomel and salts. Their effect may often be enhanced by the administration of ten grains of Dover's or Tully's powder or by a few doses of $\frac{1}{12}$ to $\frac{1}{6}$ of heroin which not only prevent the added irritation of frequent cough but seem to modify the condition in a gratifying manner. Free perspiration may be induced by some hot drink, but in such a case great caution must be exercised against subsequent exposure and checking of the perspiration.

Individuals of feeble resisting power should be kept in a warm room (68° to 72° F.) and the apartment should at the same time be well ventilated yet without subjecting the patient to the risk of a draught. In the case of young children the atmosphere of the nursery may be impregnated with steam. It is an excellent plan to apply a poultice of mustard and flaxseed meal to the chests of these little ones and afterwards to envelop them in a cotton jacket.

A dose of castor oil or calomel, bromide of soda to allay cough and a *few* drops of aconite may all be of service in lessening the initial inflammation. In all cases I attach much importance to the early use of a cathartic, counter-irritation and other derivative measures.

An acute exacerbation of a chronic bronchitis is to be treated in the same manner, that is, by measures intended to cause a derivation of blood from the actively hyperæmic mucous membrane. It is often quite remarkable how great is the relief from tightness of the chest and dull substernal pain that follows the action of a mercurial cathartic. If this be followed by a counter-irritant and by remedies to produce relaxation of the skin and perspiration in conjunction with such remedies as quiet the frequent, dry, irritating cough of acute congestion, the severity of the attack is lessened if not actually cut short.

General Therapeutics of Acute Bronchitis. — These must vary in detail according to the ætiological characters of the case, the age, strength and other conditions of the patient. The object of management is the same, however, namely, the removal of active congestion and the promotion of free and easy secretion of mucus. The former object is likely to be attained to a greater or less extent by the simple measures just proposed and in most previously healthy persons the second may be achieved by the administration of an expectorant mixture. Cases are met with every now and then however in which local and constitutional symptoms are so intense as to require careful attention and no small amount of therapeutic skill. This is particularly true of children and of old or delicate persons, especially if they have heart disease. It is likewise the case in the specific bronchitis of acute infectious disorders.

Sedatives.—The necessity for remedies of this class depends upon the frequency and intensity of the cough. When this is a prominent symptom it must be relieved, since, if not checked, it will surely aggravate the congestion and increase the patient's pain and general discomfort. If there is no special contra-indication an opiate in some form may be ordered. Dover's or Tully's powder at bedtime the first night sometimes answers admirably. In other instances $\frac{1}{12}$ to $\frac{1}{6}$ of morphine may be given along with each dose of the expectorant selected.

My preference is for heroin or codeine since, as a rule, they cause less disturbance of the system than does opium or morphine. One twelfth of a grain of heroin every six to eight hours is usually enough, but if a larger dose at shorter intervals is required it may occasion headache and nausea. In such an event codeine ($\frac{1}{4}$ to $\frac{1}{2}$ grain) may be prescribed two or three times in twenty-four hours. In all cases these remedies should be ordered in as small doses and at as long intervals as possible, and for no longer a time than is positively necessary on account of the urgency of the cough.

In the case of children or even of adults when cough is not especially troublesome it may suffice to prescribe a bromide either alone or in combination with dilute hydrocyanic acid, tincture of gelsemium, sweet spirits of nitre or ipecac. When such combinations are tried it will be found that dilute hydrobromic acid answers the purpose very well; e. g.:

℞ Acid. hydrobrom. dil.	10.0
Tr. Gelsem.	5.0
Spts. Etheris nit	30.0
Aq. q. s. ad	65.0

M. Sig.: One drachm in water every four hours.

or

℞ Acid. hydrobrom. dil.	10.0
Acid. hydrocyanic. dil.	2.0 to 5.0
Spts. Etheris nit. q. s. ad	65.0

M. Sig.: One drachm in water every four hours.

If the syrup of wine or ipecac be ordered in place of the nitre, as may be advisable in many cases, it is well to remember that since it is of itself an emetic it enhances the depressing effect of the dilute prussic and hydrobromic acids and the doses of all should be small. It is far easier to increase than to diminish the dosage of remedies whenever a change is necessary. Tincture of gelsemium is of service when cough is spasmodic or convulsive and substernal pain is unusually pronounced.

Hydrotherapy.—This is a therapeutic measure of great utility in acute pulmonary inflammations and hence applicable to the treatment of acute bronchitis, especially when fever is marked and cough is frequent, irritating and uncontrollable. It is especially praised by German authors as Winternitz and is generally employed in the form of the Priessnitz Cross-bandage. This consists of a strip of cheesecloth or thin muslin about eight feet in length and so folded together as to be of three thicknesses eight inches in width. This strip is rolled up, wet in cold water, and compressed so as not to drip. It is then applied to the thorax in the following manner described by Winternitz.

"Beginning at the left axilla it is carried diagonally over the right shoulder, reversed and brought back again to the left axilla over the back. It is then carried right across the chest to the right axilla, and thence upward to the left shoulder, to be again reversed and brought back to the starting point until the end is reached, covering as much of the chest as possible" (Fig. 4). Another dry flannel bandage is now passed about the chest in the same manner so as to completely cover the wet one, and these should then be covered by a jacket of oiled silk. The bandages may be fastened by safety-pins or tapes sewed into the ends.

Such wet applications are called stimulating, but in fact are a means of subjecting the skin of the thorax to a continuous warm vapor bath, since the heat of the body quickly changes the originally cold to a warm bandage. Lauder Brunton suggests that the *modus operandi* of this Priessnitz compress is to cause circulation in the vessels of that part of the skin to which the bandage is applied and thereby produce a contraction of the vessels of the internal parts corresponding to the particular cutaneous region.

The beneficial influence which is thus exerted upon the bronchial nerves and mucous membrane is shown by the mitigation if not the cessation of cough. At the same time the bronchial secretions are diluted and rendered easier of expectoration. Consequently, such a "stimulating" cross-bandage may quite appropriately be employed in the sedative treatment of the initial or early symptoms of acute bronchitis, whether simple or specific.

I have known such a compress to afford marked amelioration of both the fever and cough attending a bronchitis of tuberculous nature. As suitable cloth was not available, a jacket was cut out of an old comforter as follows: A portion of the comforter was taken of about the width and twice the length of the chest; this was folded together in the middle and in the center of the doubled edge a hole was cut of about the size of the neck; by making a slit down in front this aperture was then enlarged so as to admit the head (Fig. 5). This jacket was then wet in cold water and wrung out, slipped over the head and its open sides brought together and pinned. Over this rough compress was put an old flannel sheet in the form of a cross-bandage. The effect of this application was highly gratifying; the chest first became moist and during the night the entire body became bathed in a copious perspiration, yet without the young man feeling

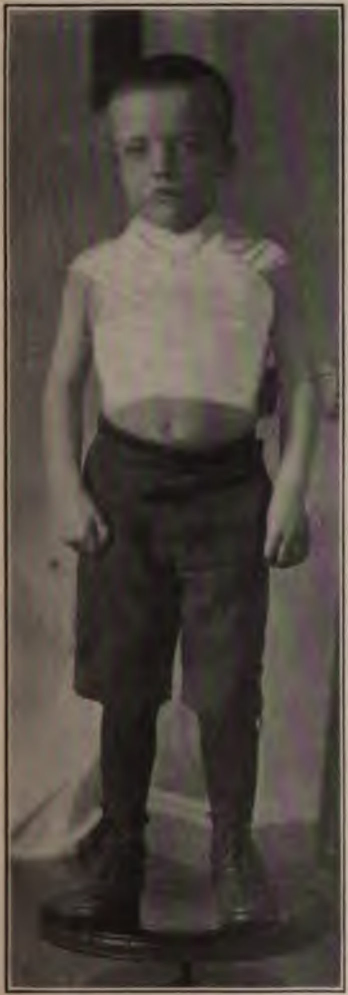


FIG. 4.—Priessnitz bandage applied.

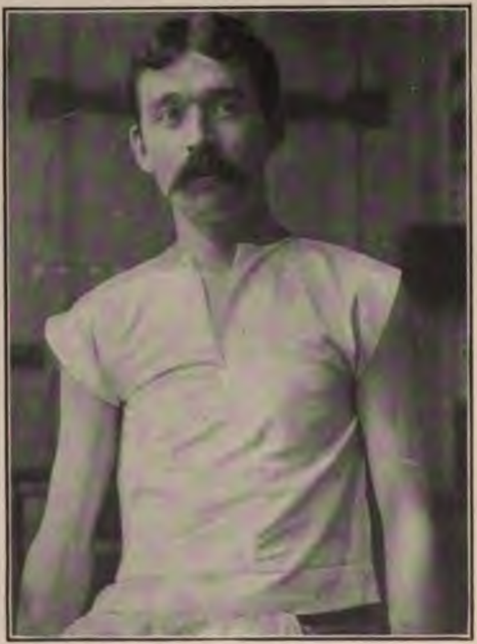


FIG. 5.—Jacket for cold compress.

chilled in the slightest. The next morning his fever was two degrees lower, his cough was less frequent, wholly without pain, and his expectoration was easy. Although the bronchitis was not gone, the symptoms were greatly ameliorated.

Inhalations.—Theoretically an acute specific bronchitis should be successfully treated by the inhalation of antiseptic and soothing remedies. Practically, however, it is not easy to cause the inhalation of antiseptics in sufficient strength to destroy the bacteria responsible for the bronchitis. Nevertheless adults may make frequent use of steam atomizers filled with an aqueous solution of some not too irritating germicidal remedy such as compound tincture of benzoin, boric acid, creosote or guaiacol. If the solution proves irritating it may be replaced by ordinary limewater which, if it cannot antagonize the organisms present in the trachea and bronchi, will at least soothe the mucosa and liquefy the secretions.

Perhaps a still more practicable means of introducing volatile antiseptic oils is a globe nebulizer which throws oleaginous solutions in the form of a fine fog that can be deeply inhaled and which enables one to breathe a far stronger percentage of the medicament without causing irritation. Menthol, eucalyptol, white-pine extract and oil of cloves are some of the remedies that may thus be tried dissolved in benzoinol to which cocaine or opium may be added if cough is very troublesome. If such an inhalation is not actually antiseptic and I frankly confess my skepticism, it is not likely to do harm but, on the contrary, is quite apt to prove soothing and acceptable to the patient.

In the case of babies and very young children the atmosphere of the nursery may be impregnated with steam from a kettle containing some such antiseptic as creosote. Some practitioners are in the habit of ordering a Cresoline Lamp, but it has always seemed to me necessary that the atmosphere be charged with the vapor of water as well as of the remedy since a moist atmosphere like an oleaginous solution is soothing to the inflamed air-passages.

Expectorants.—Unfortunately our knowledge of bacteriology has not yet enabled us to devise a certain means of combating pathogenic organisms in the bronchi and lungs. Therefore, in the disease now considered we possess no more satisfactory and available mode of treatment than by expectorants. There is a large assortment of such agents from which to choose, but if the course of acute nonspecific bronchitis is to be speedily influenced an expectorant remedy must be prescribed which will lessen inflammatory congestion and promote free expectoration. Such a remedy favors the expulsion of micro-organisms along with the secretions. Accordingly the practitioner should select one of the so-called nauseating or sedative expectorants.

In the early stage of acute bronchitis there is nothing better than hydrochlorate of apomorphin or a preparation of ipecacuanha. These both act as emetics in sufficient doses, but in less amounts they loosen cough and facilitate expectoration without disturbing the stomach. Apomorphin is a derivative of morphine and therefore in its manufacture should be carefully washed to rid it of traces of the latter drug. Although it occasions vomiting when $\frac{1}{10}$ to $\frac{1}{5}$ of a grain is injected subcutaneously, still by the mouth it can be tolerated in very much larger doses, ⅛ to ¼ and even ½ of a grain, as was originally pointed out by Sir William Murrel, and as I myself have repeatedly proven. It is quite safe, therefore, to prescribe for an adult ⅛ of Merck's apomorphin in syrup of lactucarium or malt extract to conceal its bitter taste; or, if more convenient, in capsules either with or without codeine or heroin as the case may require. Such a dose may be taken every three or four hours.

Syrup of ipecac is an excellent expectorant in these early cases, especially for children. To promote a speedy loosening of the cough it should be given to the verge of exciting nausea, i. e. 15 drops for an adult every two to three hours until the pulse grows soft and full and the skin moist. Thereupon the dose may be lessened or the intervals between its administration lengthened. Even should emesis occur, this will prove beneficial rather than harmful, unless, of course, the patient be feeble or aged. Administered in lemon juice or dilute hydrobromic acid it makes an agreeable cough mixture; but it is well to bear in mind that the hydrobromic acid seems to intensify the nauseating action of the ipecac. The syrup or wine of ipecac may also be inhaled from a steam atomizer and prove highly efficient.

Tartar emetic or other preparations of antimony and even tincture of lobelia will act in the same manner as the remedies just mentioned, but are powerful and harsh in action and therefore are less desirable for the treatment of acute bronchitis. There is another agent, the fluid extract of cocillana, which, in doses of 5 to 15 drops every four hours, acts similarly to ipecac in promoting a free secretion of bronchial mucus. Its bitter taste recommends it for those individuals who object to a sweet medicine or who complain of anorexia.

Syrup of squills *is not admissible* in the inflammatory stage of acute bronchitis because so stimulating to the mucous follicles and so irritating to mucous membranes in general as to maintain or actually increase the active congestion attending the early stage and therefore to augment the substernal pain and oppression. Even the compound syrup of squills, although containing tartar emetic, is not to be recommended at this time, since one of its ingredients is senega on account of which hive syrup becomes more suited to the subacute or chronic form of bronchitis.

In most cases of simple acute bronchitis it will be found that an expectorant remedy of the character already indicated enables the individual to get the best of his cough and expectoration in a week or so. Occasionally the condition tends to drag on in a way that brings it into the category of a subacute or even chronic bronchial catarrh. This is particularly true of children who display a catarrhal tendency, or of adults who are naturally feeble or debilitated by cardio-vascular or renal disease. In all such the management now becomes that of the chronic form, at least in respect to the kind of expectorants; and to that portion of this chapter the reader is referred.

Tonics.—Many times, and especially in patients recovering from a specific bronchitis, e. g. in children after measles or whooping-cough and in adults after influenza, it is well to prescribe a tonic. For young children nothing is better than cod-liver oil either alone or in combination with malt or hypophosphites. For adults it may be better to order some preparation of iron with arsenic and perhaps strychnine. If the heart should be feeble digitalis or one of its compeers is proper.

Change of climate for a while may often be necessary to a complete recovery and is always beneficial, especially if the climate selected is soft and balmy. The subject of climate in the treatment of bronchitis will be considered later on.

Local treatment of the throat and nares is often indispensable before a patient can entirely lose his cough. This is so because the bronchitis was preceded by an acute catarrh of the upper respiratory tract. Indeed, it is very common for a congested and catarrhal state of the pharynx or larynx to be responsible for the continuance of cough and expectoration long after the bronchitis as such has yielded to the remedies appropriate to that condition. On this account the nose and throat of every patient should receive attention and be suitably treated if they are not in a satisfactory state.

Management of Chronic Bronchitis.—Few diseases of a chronic nature require more skillful management than does the one we are now considering, and anyone who sees much of the complaint soon becomes convinced of the futility of a merely medicinal form of treatment. Accordingly the hygienic management will receive particular consideration later on and it will there be shown that hygiene and climate are of utmost importance. It is thought best at first, however, to take up the subject of medicaments since the circumstances of many patients necessitate main reliance on medicinal treatment.

Expectorants.—This is a class of remedies which have been found by experience to be of great value. Their number is legion and their therapeutic action is diverse. In some instances this seems fairly well determined, while in others the physiological action is largely conjectural and rests mainly upon empiricism. Consequently, it may conduce to a more rational therapy if the attempt is made to point out the indications for the various expectorants which experience has proven really valuable.

The more nearly a given case of chronic bronchitis approaches the acute form, as shown by substernal pain and tightness, and by a frequent, dry and unproductive cough with slight rise of the body temperature, the more definite is the call for apomorphin, ipecac, antimony, etc., that is, for agents that will lessen active congestion and facilitate expectoration. As regards the first of these I desire to repeat the statement that the dose need not be limited to $\frac{1}{8}$ of a grain but may be increased from time to time until a grain is taken every four hours. Only in exceptional instances have I known such an amount to occasion nausea, and this was either when given to a feeble individual or when it was taken before breakfast, or when it was intensified by another drug of similar action.

The so-called dry bronchitis when not due to an acute exacerbation of the malady has usually been favorably influenced by tartar emetic or lobelia in conjunction with iodide of potassium, or by the iodide together with the compound syrup of squills. The initial dosage should be small and increased as the susceptibility of the patient is ascertained. In some cases apomorphin has acted well. In this class of cases good results may sometimes be obtained by the administration of the fluid extract of jaborandi or its active principle, pilocarpine. It seems as though the secretion of bronchial mucus is favored by any drug which relaxes the vascular system and tends to produce perspiration. In numerous cases with arterial sclerosis and a dry irritating cough of congestion I have known a brisk mercurial cathartic to afford much relief.

Cases in which the expectoration is thick and composed largely of mucine generally do well on chloride of ammonium in conjunction with tincture of lobelia, fluid extract of grindelia robusta or iodide of potash, according to the difficulty of expectoration and tightness of the cough. A favorite prescription of mine formerly was

℞ Amm. chlorid.	5.0
Tr. Lobel.	10.0
Ext. Grind. rob.	fl. 15 to 20.0
Syr. Glycyrrhiz. q. s. ad	65.0

M. Sig.: A teaspoonful every four hours.

If the chest contains râles, whether solely mucous or partly dry, I have come to rely of late upon terpin hydrate which is ordered in capsules and in large doses. Small amounts of 2 grains are of little if any use, but when taken in 5-grain doses four to six times daily this remedy generally clears up the râles and diminishes expectoration in a most gratifying manner. In some cases apomorphin may be combined with the terpin hydrate, especially when cough is frequent and severe. In many cases, particularly when there is an asthmatic tendency, hydriodic acid will prove highly beneficial in association with the terpin but not in the same mixture. The two remedies may alternate, each being taken every four hours.

In concluding this portion of my subject I desire to condemn in strong terms the habit of prescribing proprietary cough mixtures which in many instances are regular "shotgun" combinations. The practitioner should prescribe in each instance with a view to meeting the indication as far as possible and should vary his combinations from time to time, as the condition changes. This is manifestly impossible if ready-made cough mixtures are prescribed.

Inhalations.—There are so many objections to the internal administration of expectorants, not the least of which is derangement of the stomach, that men of such wide experience as William Murrell have come to rely upon local medication in the form of inhalations. The remedy with which he and Ringer first experimented and from which excellent results were obtained was ipecacuanha wine. The apparatus employed was a simple double-bulb atomizer which would throw a continuous spray, and the strength of the solution was 1 part of the wine to 2 of water. At first patients are apt to arch up the tongue in such a manner, Murrell says, as to throw the solution against the roof of the mouth instead of inhaling it deeply into the trachea and bronchi as is requisite if benefit is to be derived.

If the spray collects in the mouth it is likely to be swallowed and occasion nausea. When, on the other hand, the patients learn to breathe it in deeply, remarkable amelioration of symptoms results. Murrell found that dyspnœa was the first symptom to abate, so that patients were often able to walk home after a single treatment with almost no shortness of breath. Expectoration is likely to be increased for the first few days but afterwards is appreciably lessened. This treatment is especially beneficial therefore in cases of asthmatic bronchitis. This remedy may be inhaled by means of the ordinary steam atomizer.

The second substance which Murrell found of service by inhalation was pure terebene often used as an internal remedy. It may be sprayed into the deeper portions of the respiratory tract as an emulsion, but it may be advantageously inhaled by means of the ordinary perforated zinc respirator. This is an inexpensive device consisting of a muzzle of perforated zinc having a sponge attached in front and being large enough to fit over both nose and mouth and secured to the face by means of a string passing about the head. (See Fig. 6.) The terebene is dropped freely on the sponge and the respirator is then worn for hours together or quite continuously if desired. As this remedy prevents fermentation and is a deodorizer in consequence, like oil of eucalyptus, it is of special service in the treatment of fœtid bronchitis. Murrell found it beneficial in other forms of chronic bronchial catarrh by effecting a diminution of both cough and expectoration. It does not exert especial influence, however, upon the asthmatic form as do inhalations of ipecac. Terebene may be inhaled by means of a globe nebulizer in solution of any required strength, 25 per cent or more, in albolene or respirol.

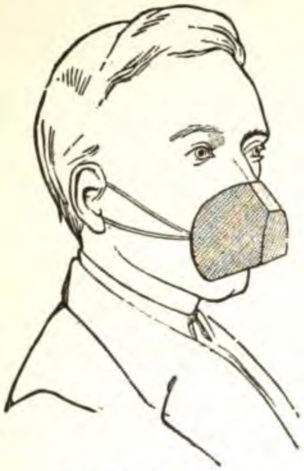


Fig. 6.—Respirator.

The inhalation of the nascent chloride of ammonia is also praised by Murrell in unstinted terms. The best and at the same time the simplest apparatus is the Bereker inhaler which consists of three bottles with connecting tubes. In the first bottle is placed hydrochloric acid, in the second a mixture of ammonia and water while in the third, which is connected with the others by means of a three-way tube, is plain water. Hence this serves as a wash-bottle for the purpose of separating any trace of acid or ammonium from the vapor.

The patient inhales the fumes of the nascent ammonium chloride through a rubber tube of sufficient length to admit of his standing erect and drawing the vapor deeply into the lungs. As free ammonia proves intolerably irritating to the air-passages this must be prevented. Accordingly it is advised that a few drops of acetic acid be put in the wash-bottle for the purpose of intercepting any traces of free ammonium that may not be held in the water. A few drops of tincture of litmus should also be added to the water to enable one to tell by the red color that the solution remains acid. So long as the water remains red there is no danger of ammonia coming over, and when the red hue changes to blue more acetic acid may be added.

Murrell states that the fumes of ammonium chloride may thus be inhaled alone or may be used as a vehicle for the administration of other agents as pure terebene. A few drops of this remedy are poured on a pledget of cotton which is then dropped into the water of the wash-bottle; its vapor then becomes combined with that of the ammonium muriate and enhances the beneficial effect of the latter.

If sufferers from chronic bronchitis are carefully instructed in the management of this apparatus and are taught how to breathe effectively, this form of treatment is said to accomplish results that are unequaled by any other mode of management. It is certainly very simple and worthy of trial, but the practitioner must take care that the patient fully understands how to avoid the injurious effects sure to result from the inhalation of free ammonium. This remedy facilitates expectoration and is said actually to bring about a cure in obstinate cases of chronic bronchitis.

Powerful Expiratory Efforts as an Aid to Expectoration.—Cough is a reflex act which may be excited by irritation elsewhere than in the respiratory passages, but in bronchitis it is generally aroused by the presence of mucus in the trachea or large bronchi. Cough is therefore the expression of an attempt to dislodge and expel the mucus. If the secretion is loose it comes up with ease but if it adheres tenaciously to the mucosa it is eliminated with difficulty. Under such circumstances patients cough long and hard before obtaining relief, in many instances not knowing how to cough effectively.

To be efficient in expelling viscid mucus, cough must be assisted by powerful expiratory efforts. Accordingly Lenhartz instructs his patients how to perform this act in the most effective manner as follows:

The bronchitic is told to stand up, and then to take a dĕep inspiration while raising his arms laterally to a level with his shoulders, that by so doing he may force in sufficient air to loosen up the phlegm. The next instant, as the patient begins to cough he aids expiration by bending his knees and bringing his arms forcibly downward and forward. In this manner great compression is exerted upon the thorax and the previously loosened secretions are driven powerfully out through the glottis.

If the patient is not strong enough to endure so energetic a movement of the arms, he is instructed to merely lower them during expiration without the forward motion. If, on the contrary, he is equal to it he is told to increase the effectiveness of the arm-movement by holding a 6-pound weight in each hand.

We are assured by Lenhartz that this simple device often proves of valuable service in the expulsion of copious, tenacious mucus that otherwise would require prolonged tussive efforts. If the desire to cough is owing to hyperæmia with but little if any mucus, such powerful efforts are useless if not harmful, and the patient is impressed with the importance of restraining his inclination to cough as long as possible.

General Hygiene.—It goes without saying that if a sufferer from chronic bronchitis is to be favorably influenced by therapeutic measures of any sort he must attend strictly to all matters of hygiene. In particular, he must avoid so far as possible whatever tends to maintain or aggravate his disease. These influences have been pointed out in such detail under the subject of ætiology that no more is here required than an allusion to them. In addition every attempt possible should be made to improve the state of the general health since the tendency of chronic bronchitis is to impair the health in general, and such impairment serves to fasten the disease of the air-tubes more tightly upon the individual.

Climate as a Therapeutic Agent.—It may be possible for an individual who is convalescing from an acute bronchitis to regain health without leaving his home climate, but if his symptoms persist though in a milder form despite suitable therapy there is great danger of the ultimate development of a chronic bronchial catarrh. Especially is it advisable for persons just recovering from influenza, measles, etc. to make a change of climate until their mucous membranes have been restored to a state of health.

The same recommendation applies to persons who begin to manifest a chronic winter cough or who find that with each recurring Fall they take cold more easily. If they are well on in years and have signs of cardio-vascular and renal degeneration, emphysema or an asthmatic tendency they ought by all means to seek a suitable climate as soon as the weather at home becomes inclement.

Furthermore, the patient ought not to be allowed to select a resort on the recommendation of some lay friend, nor should he be merely advised by his family doctor to go to a mild climate without having the resort chosen with an intelligent view of the indications in his particular case. Comfortable accommodations and a good local practitioner are also most important and should be kept in mind in the selection of an abiding place for a winter. When Cuba and other islands of the West Indies shall have provided for the care of health-seekers from the United States, they will doubtless become the Mecca for invalids of the sort now considered.

Persons with irritable mucous membranes and a persistent dry cough usually obtain relief in a warm and moderately moist climate since it soothes the air-passages and promotes the secretion of mucus. Such climatic conditions are found in the islands of the Gulf and along the Gulf coast of our Southern States. The spot chosen must be protected, so far as possible, from the cold " northers " that sometimes sweep down into the South and carry along with them possibilities of much harm to the chronic bronchitic.

The sudden changes of temperature occasionally experienced in our Southern States furnish a strong objection to them in the minds of some persons. For such the climate of Southern California offers many attractions. It is fairly dry but along the coast is less so than in the foothills. Taken altogether it is excellent for individuals who are suffering from bronchitis and do not require a relaxing climate. It is probably more suitable to persons having abundant bronchial secretions.

It is impracticable in the limits of this work to enter into a detailed discussion of the effects of different climates and the various resorts which present these varying features. It must suffice, therefore, if the indications are laid down in a general way which should govern the choice of a health resort.

Warm, moist climates are indicated in acute conditions and in dry bronchitis or when the bronchial secretions are viscid and difficult of expectoration. A warm, dry atmosphere, on the other hand, is best when the cough is loose and the sputa are copious from large admixture of serum and pus. If emphysema is present it contra-indicates a high altitude, the actual degree of elevation permissible being determined by the severity of the emphysema. Asthmatic patients generally do not bear much of an altitude, and yet this appears to be governed in the main by the degree of the associated emphysema. As a general proposition aged persons would better remain at or near the sea level. Whatever the climate selected it must be equable and not subject to great differences between the night and the day temperatures. Lastly it must be free from dust and dust-storms.

Spas.—In Europe great value is attached to the use of natural waters and chronic bronchitics are sent to certain resorts to drink the waters and to profit by the climatic conditions which there prevail. Ems, Neuenahr, Carlsbad, Weilbach, Kissingen, Homburg, Marienbad, Vichy, are the best known. Hoffmann is of the opinion that the chief benefit comes from the ingestion of large amounts of fluid, while Lenhartz believes that positive value must be attached to their saline and alkaline ingredients. The latter author especially recommends the drinking of the waters of Kissingen, Homburg and Marienbad by persons who suffer from hyperæmia of the pharynx and digestive disorders.

At most if not all of these watering places there are inhalatoria, i. e. cabinets or chambers in which patients sit and breathe an atmosphere impregnated with the vapor of the waters. The benefit thus derived comes from the inhalation of sodium chloride which forms a large proportion of the mineral constituents of the springs, as well as from breathing the moist atmosphere. Lenhartz asserts that he has witnessed gratifying results follow such inhalations.

According to Guy Hinsdale the following springs of the United States are analogous to the German spas just mentioned.

Ems—Healing Springs, Virginia; Ojo Caliente, New Mexico; Enna Soda Springs, California; Hot Springs, North Carolina.

Carlsbad—Springdale Seltzer, Pagosa Hot Springs, Royal George Hot Springs, Idaho Hot Springs, Colorado; Bedford Magnesia Spring, Pennsylvania, partly. American Carlsbad, Washington County, Illinois.

Homburg—Solubria Spring and Glenn Springs, New York.

Kissingen—Sheboygan Mineral Well, Wisconsin; Lower Blue Lick Springs, Kentucky.

Vichy—Geyser, Saratoga, New York; Ukiah Vichy Spring, Mendocino County, Congress, California; Vichy Spring, Saratoga, New York; Ojo Caliente, New Mexico.

Our American spas are rich and varied, but have not been adequately investigated and are not so far as I am aware provided with inhalatoria for the proper treatment of chronic bronchitis. Nevertheless, in the choice of a health resort for this class of invalids the physician might very properly recommend some one of the list given above where climatic conditions are right and the waters would be likely to prove beneficial.

Treatment of Fibrinous Bronchitis.—This it must be confessed is very unsatisfactory, since we possess no means of preventing the formation of the casts or indeed of furthering their expulsion. Consequently our therapy must be largely symptomatic and palliative. When cough is so severe and prolonged as to necessitate its mitigation, we are compelled to rely upon some form or derivative of opium. Care should be exercised, however, lest the sensibility of the sufferer be too powerfully blunted since the act of coughing is essential to the elimination of the casts.

Expectorants are probably of little if any utility, but if tried should be such as belong to the emetic class. Indeed, Lenhartz mentions emetics, as indicated, while at the same time cautioning against their injudicious use. If it be decided to try the influence of emesis in expelling the mold, then the hypodermic injection of $\frac{1}{10}$ to $\frac{1}{6}$ of a grain of apomorphin is undoubtedly preferable to depressing remedies administered by the mouth.

Fowler suggests the possible utility of inhalations or intratracheal injections and cites the experience of a colleague who thought he had facilitated the expectoration of the cast in one instance by the intratracheal injection of glycerine. Limewater spray has been recommended for the removal of false membrane on the throat, and therefore this simple remedy might be tried by inhalation from a steam atomizer, it being possible that some additional good might follow the relaxing effect of the warm vapor.

Hoffmann, who indorses the use of limewater in the form of a spray, remarks upon the wisdom of treating acute plastic bronchitis in accordance with its ætiology, so far as this can be determined. Theoretically this is good advice, but with the exception of cases depending upon Loeffler's bacillus we possess no certain therapeutic agent. Inhalations of creosote have been recommended as have been other drugs on the theory of their exerting an antibacterial influence upon the organisms concerned in the ætiology of the affection.

Potassium iodide is the remedy most generally prescribed in this disease and in a few cases it appears to have been of value. It may be given during the attack to facilitate the expulsion of the casts and in the intervals to prevent their formation. The dosage does not need to be large, ten to fifteen grains thrice daily being sufficient.

The general management is highly important, since measures that improve the health undoubtedly tend to lessen the liability to the affection. If, as stated by Hoffmann, tuberculosis is responsible for a large proportion of the observed cases, then the necessity for careful dietetic and hygienic management becomes at once apparent. Consequently, all that has been said on the climatic and general treatment of chronic bronchitis applies also to this form.

CHAPTER VI

BRONCHIECTASIS

THIS term signifies a dilatation of the air-tube which, as will be shown in considering the morbid anatomy, is divisible into a number of varieties. The condition here described scarcely constitutes a clinical entity *per se* since it is associated with some other primary or predisposing affection whose symptoms disguise or conceal those of the bronchiectasis. There are cases, however, in which the bronchial dilatation declares itself by quite definite and distinctive clinical features which permit of its diagnosis and hence the designation of the dilatation as the chief clinical entity.

Pathogenesis.—If one considers that the predisposing affections occur with infinitely greater frequency than does bronchiectasis, it is at once plain that aside from these we must look for other factors that act as determining causes. In other words we must endeavor to explain why the bronchial tubes become dilated in some cases and not in others.

Accordingly numerous theories have been advanced, and although writers are agreed on some points, they are not yet in accord on all. Laennec propounded the hypothesis that dilatation of the bronchi is the result of increased intrabronchial air-pressure, while Corrigan expressed the opinion, afterwards so ably espoused by Sir Andrew Clark, that the determining cause in very many cases is traction on the bronchial wall from without, through the contraction of fibrous bands as will be more fully considered later on.

The influence of increased intrabronchial air-pressure is now generally accepted, but inasmuch as the pressure of air within the bronchi is raised to a high point by a great variety of conditions, notably by coughing, crying, singing, etc., yet without the production of bronchiectasis, something more than this is necessary.

This additional factor is generally conceded to be an inflammatory change by which the elasticity of the bronchial wall, and hence its resistance, is diminished. If ordinary inflammation were thus capable of injuriously disturbing the resistance of the tube, then the association of bronchitis and bronchiectasis ought, as Hoffmann urges, to be very common, which is not the case. Consequently, Hoffmann declares, the inflammation must be a specially severe one, and such is found in the bronchitis of measles, whooping-cough, diphtheria and the broncho-pneumonia of influenza and typhoid which according to Marfan is a particularly frequent cause of bronchiectasis.

Hoffmann's argumentation seems to me very strong. He calls attention to the fact that when expiratory effort is made against the closed glottis air-pressure is raised to 80 mm. of mercury, whereas during ordinary respiration the pressure within the trachea is but 2 or 3 millimeters. Such an abnormal degree of pressure is sustained by the healthy bronchi without injury whenever an individual coughs or sings and the like. During such an expiratory effort the entire bronchial tree is distended and may be said to be in a state of temporary bronchiectasis from which it recovers immediately in consequence of its elasticity. If the ability of any portion of an air-tube to withstand such pressure is impaired, then such limited portion yields permanently to the distending force and bronchiectasis is the result.

Diminished resistance at one or more points is not the only requisite, however, according to Hoffmann; for, if it were, dilatation of the bronchi would be far more frequent than it is. There must also be a condition which leads to abnormal augmentation of air-pressure, and this he finds in stenosis. He admits that narrowing of the lumen of the bronchus is not discovered in every instance of bronchiectasis, but this in his opinion is due in some cases to failure in recognizing stenosis, while in others there is an absence of still other conditions deemed by him essential.

Such necessary requirements are (1) inflammation and (2) a relatively free ingress of air during inspiration as compared with the difficult egress during expiration, in consequence of which the air contained in the tube peripheral to the constriction is confined under high pressure. In support of his contention Hoffmann cites Lichtheim's experiments by which the production of bronchial stenosis in dogs was followed by the development of bronchiectasis and the dilated tube was found more or less filled with thick secretion.

He argues that in consequence of the entrance of germs with the air the contents of the tube beyond the seat of stenosis become infected with resulting inflammation of the bronchial wall. When infection does not take place, stenosis leads to atelectasis of the lobules supplied by the tube without dilatation of the bronchus. In this manner the first essential, i. e. inflammatory impairment of resistance, is fulfilled.

The collection of thick tenacious secretion at the distal side of the stenosis furnishes the second requirement, since it acts as a valve by permitting air to be driven in with the positive pressure of inspiration but prevents the escape of air during the lessened pressure of expiration accomplished, as this latter is, by the elastic recoil merely of the lung.

According to this view he accounts for the formation of bronchiectases which have been found in cases of aortic aneurysm that has partially occluded a bronchus, in cases of syphilitic bronchial stenosis in which dilatation of the air-tubes is a very common occurrence, and even in cases of pulmonary tuberculosis. In the last-mentioned disease he sees in the development of the tubercle itself in the wall of a bronchiole a possible cause of stenosis and hence of dilatation. In fibroid phthisis he recognizes an additional factor, namely, traction on the bronchial wall by the fibrous tissue. In bronchopneumonia, furthermore, Hoffmann believes that the inflammatory tumefaction of the bronchial mucosa, aided by the secretions, is capable of setting up stenosis and thus predisposes to the development of bronchiectasis.

Lenhartz agrees with Hoffmann in attributing ætiological imp bronchial stenosis when this is due, e. g., to cicatricial contraction, l Hoffmann goes too far in his attempt to find narrowing of the lume essential in all cases. He thinks inflammation of a limited part of and increased air-pressure therein are sufficient ætiological facto instances.

Fowler recognizes three intrinsic causes, (1) acute and chronic (2) bronchial stenosis and (3) obstruction of a bronchus as by a fo All these conditions favor distention of the tube by violent expirat during cough, but this alone would not suffice, and therefore he al there must be loss of elasticity and of muscular tone from inflamn bronchitis " retained secretion, which is no longer permeable to air, a better *point d'appui* for such a force " (the distending force of effort) " than air contained in the tubes, and secretion may be retai the lung tissue beyond has undergone collapse. A deficient entry of a part of a lung, by producing incomplete expansion of the alveoli an tive support of the bronchi, would necessarily favor dilatation of but such a condition, although commonly present in cases of bro cannot be regarded as essential."

In bronchial stenosis Fowler regards the three following caus tributing to the result: " (1) Accumulation of secretion in the tu obstruction of the bronchioles and collapse of the tributary area. (in the bronchi and peribronchial tissues diminish the power of re pressure from within. (3) Fibrous thickening of the interlobular alveolar connective tissue, and of the lung as a result of inflamm to contraction of the surrounding tissue."

The foregoing quotations from such well-known writers show tha authors agree in the main regarding the causes of bronchiectasis differ in the matter of the contributing factors. The essentials are stated, loss of elastic resistance on the part of the bronchial wall an expiratory air-pressure.

There now remain to be considered the cases of obliterative chronic interstitial pneumonia, fibroid phthisis, etc., in which the d cause is believed to be traction on the bronchial wall. It was t cases that led Corrigan to express the view afterwards adopted by I Lebert and others, that in consequence of the contraction of fib which extends from the pleura inward or forms within the lung a the bronchi and is in fact an indurative process, the walls of the ai drawn outward. This tendency to dilatation is furthermore aug air-pressure during inspiration.

The coexistence of bronchial dilatation with cirrhotic changes i is not constant, Bastian having found no evidence of bronchiectasis of 30 cases collected by him. It is said to be the early cases in wh tion of the bronchi is not discovered. The explanation seems to be shrinkage in the size of the lung must occur before the air-tubes sufficient traction to undergo stretching. At first the contraction of tissue is at the expense of the chest-wall, pulmonary parenchyma num and diaphragm, which are all drawn inward and upward so as to produce great diminution in the diameter of the affected half of the thorax. At length however there comes a time when these structures cannot yield any further and, contraction of the new-formed tissue still continuing, traction is exerted on the bronchial walls with resulting dilatation.

Nearly all writers attribute more or less influence to this factor of traction in certain cases, but Hoffmann believes that even in such cases there is also the factor of bronchial stenosis produced by the fibrous tissue.

Fowler, on the contrary, expresses the opinion that fibrosis of the lung tends rather to obliteration than to dilatation of the tubes. "That they are not completely obliterated is due partly to the dilating force of the chronic cough, which is commonly present, and partly to the fact that when, by the aid of such changes as approximation and overlapping of the ribs, curvature of the spine, displacement of organs and of the mediastinum, and enlargement of the opposite lung, every possible compensation has been made, the equilibrium between the various forces is obtained before the cavities in the lung have been completely obliterated."

Ætiology.—What has been said concerning the pathogenesis of this affection renders superfluous any extended discussion of its ætiology. Bronchial dilatation is a comparatively frequent discovery in the *post-mortem* room and is associated with changes in the air-tubes, the parenchyma or pleura which, during life, were perhaps the only changes recognized. The nature and variety of these primary affections are well shown by the annexed table quoted from Fowler and Godlee and compiled from the autopsy records of Brompton Hospital from 1887 to 1894. The diseases herein mentioned render unnecessary any especial remarks regarding their ætiological influence, particularly when viewed in connection with what was said under pathogenesis.

Acute bronchitis	1
Chronic bronchitis	7
Syphilitic stenosis of the bronchi	1
Pressure on bronchi by mediastinal growth or an aneurysm	1
Foreign bodies in the bronchi	2
Bronchitis and asthma	1
Broncho-pneumonia	5
Lobar pneumonia	2
Chronic pneumonia and cirrhosis of the lung	5
Pulmonary tuberculosis	4
Pleurisy and empyema	3
Hydatid cyst of the kidney	1
Spinal curvature	2
Total	35

Acute Bronchitis.—The association of bronchial dilatation with acute bronchitis is seen most often in children, although it may occur in adults. The bronchitis is not the simple or nonspecific variety due to extension from above, but is the form known as secondary or specific, depending upon pertussis, measles, diphtheria, etc. Biermer speaks of it as occurring in connection with capillary bronchitis and it is stated by Marfin to be frequent in broncho-pneumonia, a statement borne out by the ta above.

It is believed by some writers that dilatation of the tubes in c with bronchitis is not always a permanent condition, but that it (after the subsidence of the inflammatory process because the elastic walls has not been seriously impaired. This will be referred to aga cussing the influence of some of the other associated diseases in the p of bronchiectasis.

Age exerts no direct ætiological influence since the disease now (may be found at any time of life from that of early childhood t(Bronchial dilatation is however most frequent during middle age, only because the diseases with which it is associated are most freque this period of life.

Sex.—The same statement may be made concerning sex as regarding age. Although men appear to suffer from this affection n than do females, 29 out of the 35 cases quoted above having been still such a preponderance of males is not to be ascribed to sex *per* the circumstance that men are more liable than women to the pr diseases already mentioned.

Other ætiological factors, as heredity (Grainger Stewart), ana turbances of the general health, etc., if they exert any influence at only very remotely by preparing the way for the development of th of pleura, lungs or bronchi which are generally recognized as predi bronchiectasis.

Morbid Anatomy.—Two varieties of bronchiectasis are gen scribed, the cylindrical and saccular. The term fusiform is ap subvariety or modification of the cylindrical. These last are unifo tions which involve the entire diameter of the tube, while saccula chiectases are globular pouchings very like a sacculated aneurysm. the air-tubes may become dilated, but the large bronchi are less affected than are those of medium caliber.

The number is also variable. There may be but a single bro and when so it is usually sacculated, or the dilatations may be so especially when of the cylindrical variety, as to have converted entire lung into a series of cavities (Fig. 7) (the so-called tur Such is the condition observed in the congenital bronchiectasis of c writers. A cross-section of a lung which is the seat of cylindrical tases presents an appearance suggestive of a slice of Swiss cheese o sponge. A single bronchus may display a series of beadlike dilatati give it a resemblance to a rosary, the bronchiectases of this kind be times styled moniliform.

Bronchiectases may exist in any part of one or both lungs but often unilateral and at the base. If associated with fibroid phthisi apt to be at the upper portion and of the sacculated variety, whenc ficulty of always distinguishing them from tuberculous vomicæ, pa if there is but one cavity and its wall has undergone ulceration.

chiectasis, especially when saccular, generally contains purulent material which, by reason of decomposition, is usually horribly fœtid.

The walls of the dilated tubes display changes of a chronic nature. The mucous membrane shows the changes of long standing bronchitis, being in some places thickened and congested, in others atrophied, ulcerated or even calcified. The elastic and muscular layers may be atrophied or the wall may have become thin and fibrous like scar-tissue. Peripheral to the point of dilatation the bronchus is occluded and shrunken, and in some instances the tube has also become sealed at the proximal side of the ectasis so that the cavity is shut off, and its contents are thickened and gelatinous or calcified.

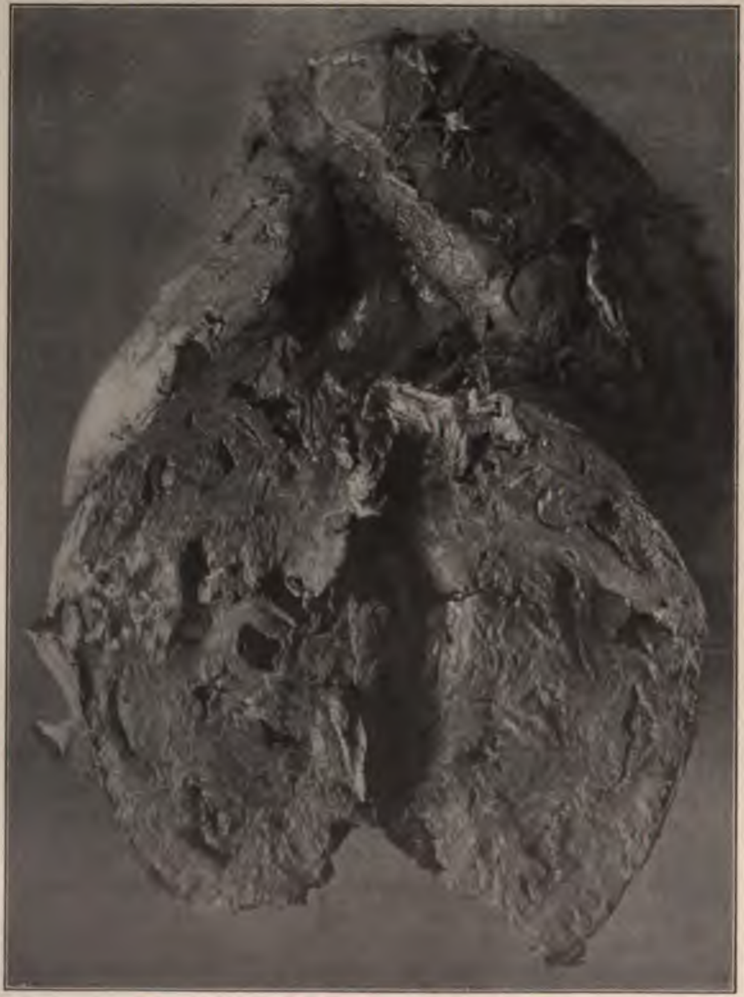


Fig. 7.—Lung from case of bronchiectasis.

In the sacculated variety it is not very uncommon to find the wall ulcerated and the surrounding tissues invaded by the same destructive process, and perhaps gangrenous.

The pulmonary parenchyma has also suffered in its integrity, usually as a primary and not a secondary condition. Thus, in a young man of twenty-five sent to me by Dr. W. S. Brown of Elgin, Illinois, who subsequently died as a result of operation, the right lower lobe consisted of a series of cylindrical bronchiectases separated by thick sæpta of fibrous tissue and was greatly reduced in size. The middle lobe also showed a few dilatations, while the upper lobe, as is usually the case, was emphysematous and occupied the major portion of the pleural cavity on that side.

Often the pleuræ are thickened and adherent, the entire lobe or even the whole lung being converted into a dense mass of fibrous tissue and bronchiectases, while the respective half of the thorax is greatly retracted and the heart displaced.

Congenital atelectasis may occur in the newborn, when it is thought due to hereditary syphilis, or, as in a few recorded cases, in adults. The lung is filled with cysts containing a serous fluid. The lobe is thus converted into a flaccid pouch which does not communicate with the bronchi and in Grawitz's case careful examination showed a system of small cavities opening into a common middle cavity and divided from each other by thin walls. From the largest of these arose secondary and even tertiary partitions, numerous recesses being thus formed. The fluid in the main sac which Grawitz thought might have been formed by a hydropic distention of a bronchus, contained nuclei and cylindrical cells, while the walls were lined with cuboidal epithelium, the uppermost of these cells being ciliated. The cyst was bounded by a small zone of embryonal pulmonary tissue and was thus united to the remainder of the lung which was healthy.

Kessler discovered a similar condition in a baby girl of five months. The upper part of the left lung was converted into a white fibrous sac into which numerous bronchial tubes opened, while the sac was connected with the hilus by a limited amount of normal pulmonary tissue. This patient had suffered from asthmatic attacks which were aggravated by the right lateral decubitus. Another very similar state of things was found in a five to six months' fœtus by Meyer. Fraenkel describes a like condition in a man of twenty-five years who had suffered from an asthmatic complaint.

There is another class of cases also regarded by Grawitz as having been congenital. In these there are many cysts located in the bronchi of the third and fourth order, sometimes arranged in rows, sometimes opening outward but sometimes closed. Only one lung is affected and there is an absence of pigment in the lung.

In some cases there may be a complete atrophy of an entire lung in association with bronchiectases. Four such cases were reported by Neisser in all of which the left lung was the one affected. The symptoms of dyspnœa, cough and expectoration of a bloody, sometimes fœtid, sputum, had existed intermittently since early childhood. It is worthy of special note that the diseased half of the thorax was not retracted, and Neisser believes that in congenital cases of bronchiectasis with atrophy shrinkage in the side of the chest does not occur.

The cases of one large cyst developed by dilatation of a large bronchus are termed by Grawitz universal ectasia, while the others in which multiple cysts are found he terms the telangiectatic form.

Finally, Heller has described the *atelectatic variety of bronchiectasis.* In this the cavities are separated by portions of lung tissue displaying the characters of fœtal lung and there is a great growth of cartilage in the walls of the bronchi. Ciliated epithelium is wanting, being replaced by cells of the squamous variety.

Symptoms.—In the early stage of the disease the symptoms do not vary much from those of ordinary bronchitis and consist chiefly of cough and expectoration. Later these two features become quite distinctive. There are also the symptoms due to malnutrition and toxæmia, as will be stated subsequently.

Cough.—This in well-marked cases is paroxysmal and often severe but in frequency depends upon the rapidity with which bronchial secretions collect. After the emptying of the bronchiectatic tubes cough subsides and remains quiet until the cavities again require emptying. The interval of quiet may last from minutes to hours. So long as the contents of the bronchiectasis are confined to the cavity they do not excite cough, but as soon as they overflow their bounds and, entering the irritable portions of the tubes above, cause increased irritation the reflex act of cough is evoked. In some instances the patient may control this symptom until some change of posture causes the secretions to flow out into the comparatively healthy bronchi above when he suddenly and spasmodically coughs and expectorates in the manner characteristic of bronchiectasis. As a rule cough is not painful but may be so when it is so violent as to occasion muscular strain.

Expectoration.—This is characterized by its abundance and the manner of its expulsion. The amount is variable in different cases but is usually large, in some instances reaching a pint or more in twenty-four hours. Trojanowsky has recorded 800 c.c. in a single day (Hoffmann).

A striking characteristic of the expectoration in many cases is the manner of its expulsion. It comes up in such quantities and suddenness as to actually gush from the mouth and even through the nose. This is said to be the case in globular dilatations especially, since the configuration favors the retention of the secretion until it attains a large amount.

Another striking feature is the effect of posture upon the expectoration. If the patient lies on the affected side he may not cough, but immediately upon turning to the opposite side, bending forward etc. he may be able to eject large amounts of sputa.

The young man from whom the specimen shown in Fig. 7 was obtained was always able to clear out his tubes of plentiful, bitterish, stinking pus, to his immense relief, by stooping forward or straining so as to force his diaphragm upward and compress the lung. My examination of his chest directly after such an act disclosed marked tympany and loud rhonchi at the right base where previously dullness and feeble breath-sounds had existed.

Fœtor of the sputa is often but not necessarily present in bronchiectasis and then depends upon decomposition of the secretions *in situ*, the same as in fœtid bronchitis. In the case just mentioned the expectoration gave forth an intensely disagreeable odor which was perceptible also in the breath. It was not like that of gangrene but had a faint resemblance to wet mortar. In some instances the sputum is said to resemble the fœtor of gangrene. Such is of course the odor when gangrene is actually present, but the fœtor may be very similar from decomposition without gangrene. In all instances the breath emits the same foul odor.

Allowed to settle in a glass vessel the sputum separates into layers of which the top one is dirty froth, the middle one is muddy-looking serum and the nethermost is composed of thick sediment. Examined with a microscope this sediment is found made up of granular *débris*, fat drops, crystals of the fatty acids, various micro-organisms and, in some instances, Dittrich's plugs. Shreds of lung tissue are not present unless the lung is also in a state of gangrene.

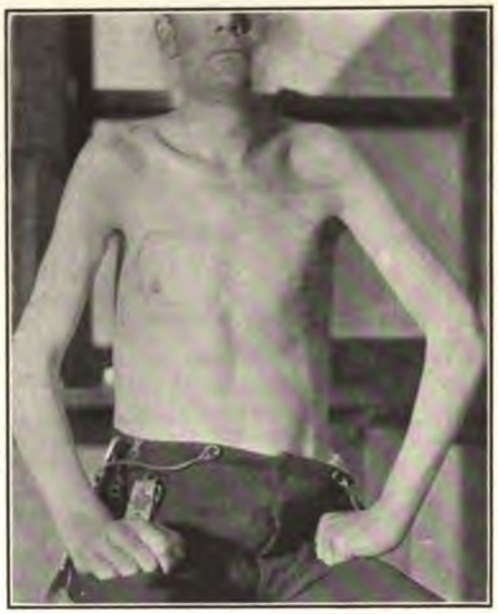


FIG. 8.—Photograph of patient with bronchiectasis.

The organisms found in the sputum are streptococci, staphylococci, rarely pneumococci, vibrios and leptothrix.

In Hoffmann's opinion a suspicion of gangrene should be entertained whenever the sputa become fœtid and of a dirty brown color. The decomposition of the secretions in the tubes is apt to be attended by fever and by other symptoms of the absorption of septic products. The young man above mentioned declared that when he could not empty his tubes he was apt to experience a slight chill, moderate fever and headache.

Dyspnœa.—This is not a customary symptom of bronchiectasis unless there be some complication as acute bronchitis, broncho-pneumonia, emphysema or secondary cardiac asthenia. There is likely to be temporary shortness of breath directly following a prolonged paroxysm of cough. Asthmatic seizures are rare and due to emphysema. A man in my service who died at Cook County Hospital suffered sorely from this type of breathing during the few hours intervening between his admission and his death. The clinical findings were those of bronchitis and emphysema, yet the autopsy disclosed widespread bronchiectases and fibrosis.

Hæmoptysis is rather infrequent and does not properly belong to the symptomatology of the affection under consideration. When it occurs the amount of blood expectorated is usually small; yet severe hæmorrhage may result from ulceration of the bronchial wall or the neighboring pulmonary tissue, and the consequent erosion of a vessel of considerable size. Cases have been reported in which profuse hæmoptysis has been the immediate cause of death. Fox states that this symptom is rare without the association of tuberculosis as cause of the ulceration, a view corroborated by the fact that Barth found tuberculous disease in 4 of his 7 cases of fatal hæmorrhage occurring in the course of bronchiectasis. Nevertheless, as believed and stated by Biermer and Lebert, slight hæmoptysis may arise without tubercle, in consequence of extreme injection of the capillary plexus within the bronchial mucosa. The repeated occurrence of hæmorrhage in this class of cases is attested by one of Laennec's 2 cases, that of a woman of seventy-two who had been subject to hæmoptysis at intervals for fifty-six years.

From the foregoing, it is apparent that cough and expectoration are the only essential symptoms of bronchiectasis and that such other syndromes as may be observed are accessory and owe their presence to complications or sequelæ.

Pyrexia is the most important of the secondary symptoms and is to be regarded as an indication of sepsis. It may, as already stated, be an expression of infection arising from absorption of toxines from the decomposed secretions in the bronchi; but it may result from a far more serious condition, i. e. gangrene. The fever in the former instance is not apt to be high and is of a remittent type, 100° F. to 101° F. in the A. M. and 101° F. to 102° F. in the latter part of the day. When it is due to gangrene the temperature curve is apt to show much more sudden and extreme variations.

Chills and sweatings are not pronounced in the milder grades of toxæmia but are likely to be pronounced on the occurrence of gangrene. There is no constant rule however in this regard, even when active destruction of pulmonary tissue has been added.

The symptoms just mentioned may be of short duration, a week or two, or they may persist until the patient's strength is worn out and he succumbs to the exhausting effect of the septicæmia. Recurrences of this milder form of infection may take place repeatedly during the years in which the bronchiectasis lasts. As just implied by the foregoing remark, bronchiectasis is a chronic malady in the great majority of instances. Although bronchial dilatation may develop acutely in the course of specific bronchitides and broncho-pneumonia, in which event it may disappear after subsidence of the primary affection, still bronchiectasis does not usually pursue such a course. It persists for years and exhibits a checkered course of longer or shorter duration, according to the occurrence or not of some of the complications subsequently to be noted.

Malnutrition and emaciation are inevitable in the course of time, and at length the sufferer comes to present the appearance seen in cachexia, from whatever cause. The skin becomes dry and of an ashen gray color, which, in some cases, may be modified by the cyanosis incident to the interference with pulmonary circulation.

Clubbing of the fingers and toes becomes a very frequent feature, so frequent in fact as to be almost a diagnostic point. This is a thickening of the terminal phalanges in consequence of which they assume a bulbous or rounded form very like that of a drumstick or club (Fig. 9). Not only is the end of the finger or toe rounded and swollen, but the nail is curved in a very characteristic manner, like a hawk's beak, which serves to distinguish clubbing from the increase in size of acromegaly, the nail in this affection being relatively small and flattened. When clubbed the finger-ends may also be cyanotic in consequence of capillary stasis, a condition that is probably one of the ætiological factors.

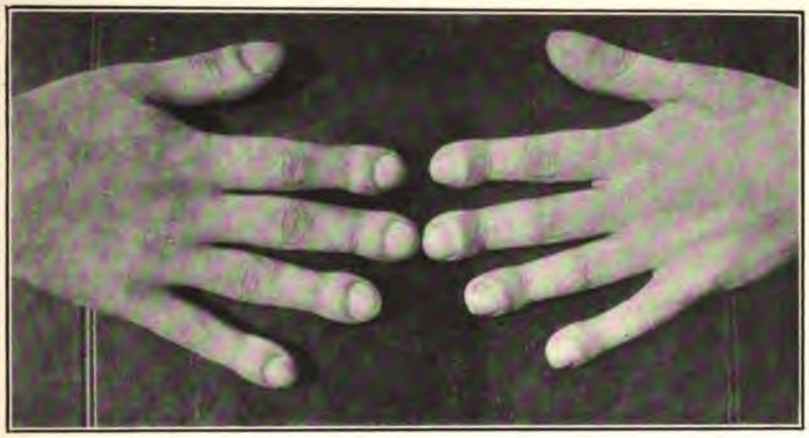


FIG. 9.—Clubbing of fingers of patient shown in Fig. 8.

The condition now described is not confined to bronchiectasis but is seen in several other chronic affections of both lungs and heart. It may be seen in emphysema with chronic bronchitis and asthma, but is more common in chronic phthisis, empyema and even in pulmonary abscess, in which latter affections it is probably the result of the absorption of toxines of suppuration. It may arise suddenly in a period of two or three weeks and it may disappear rapidly after the primary disease has been cured.

This bulbous enlargement of the fingers and toes may in some instances be owing to a hyperplasia of the osseous structures but certainly is not so in all. The abrupt development and disappearance of the clubbing proves conclusively, I think, that the swelling is really due to thickening of the soft tissues in such cases and not to a true hyperplasia. The absence of bony overgrowth is borne out by the results of X-ray examinations. In the instances in which the clubbing develops rapidly the ends of the digits may be painful and may even feel hotter than normal. I once observed this sensible increase of heat together with redness in a young woman who was suffering from subacute endocarditis.

I recall a boy in the Cook County Infirmary at Dunning who presented this clubbing of the terminal phalanges in a very marked degree, and in whom the physical signs and fœtid purulent sputa led to the opinion that the pulmonary disease was chronic bronchitis with bronchiectasis. For weeks he was in a mild septic state which led ultimately to his death. Unfortunately an autopsy could not be obtained.

Pulmonary osteo-arthropathy, another condition which occurs in bronchiectasis and in chronic pulmonary tuberculosis, may be mentioned in this connection. It was first described by Marie in 1890 and since then has been studied by many others, among whom may be cited Bamberger. Strangely enough the patient who led Marie to make his communication under the title of *Hypertrophiante pneumique* was subsequently found to have suffered from acromegaly. The affection under consideration is a thickening of the ends of the long bones of the arms and legs and of the shafts of the fingers, of the metacarpal and even the carpal bones and the corresponding bones of the feet. This thickening is a true hypertrophy in consequence of a periostitis that leads to an increase of the bone; while at the same time there is a rarefaction of the osseous tissue, with dilatation of the interstices which naturally exist in the bone, as well as increase of its medullary substance (Fraenkel). These changes may be accompanied by pains and tenderness to pressure which, arising in the affected areas of the extremity, may spread up the limb and involve the neighboring joints.

Complications.—Of those most likely to modify the course of bronchiectasis the most frequent is the sepsis which it has already been stated follows the decomposition of the contents within the dilatation. The possibilities that may result from bacterial infection of the secretion are well shown by the case reported by Thiroloix and mentioned by Hoffmann, in which were discovered malignant endocarditis, multiple hepatic abscesses and abscess of the kidney, all probably due to the staphylococcus aureus, since this organism was obtained from the bronchiectatic cavity as well as from the other lesions mentioned. It should also be borne in mind that *abscess of the brain*, usually, it is said, of the side corresponding with that of the affected lung, sometimes occurs, and that Nothnagel has recorded abscess of the spinal cord.

Arthritis, especially of the ankle and knee has been noted and was first reported by Gerhardt. It is of course but another manifestation of the infection to which these patients are exposed. Still another result of the chronic absorption from purulent secretion within the dilated tube is *amyloid degeneration of the kidneys* recorded by Trojanowsky. *Pyothorax* may also occur but when it does, it is owing to rupture of a superficially situated bronchiectasis into the pleural cavity.

Pleurisy, acute bronchitis, broncho-pneumonia, and **tuberculosis** are not infrequent complications, and may even prove terminal events. They are also attributable to infection. Lastly there are *circulatory complications* that are to be referred to mechanical interference with the circulation and right ventricle dilatation. These are dropsy, albuminuria and cyanotic induration of the liver, all of which modify the clinical picture and contribute to the fatal result.

Bronchiectasis in a somewhat different clinical picture from th is also believed by J.

Walter Carr to be of far more frequent occurrence than is generally thought or would be inferred from the literature. Carr published in the *Practitioner* for February, 1891, an account of 6 cases which he had observed in his hospital service. Kingston Fowler devotes a short chapter to this phase of the disease and frankly admits he was largely influenced to do so by Carr's paper. French writers, notably Marfan recognize the importance of the subject, but Hoffmann in *Nothnagel's System* and Lenhartz in *Ebstein's Practice* merely allude to it incidentally. In the United States it has received very little attention. Holt speaks of it only in connection with chronic bronchitis and chronic broncho-pneumonia, while Rotch scarcely mentions bronchiectasis at all.

When, therefore, men of wide experience fail to devote special consideration to bronchial dilatation in young children, the explanation must be either that they regard bronchiectasis as but a minor part of a bronchial or pulmonary condition to which the symptoms are mainly referable, or that in this country the inmates of children's hospitals are in better general health than in Europe.

The children furnishing the material for Carr's report were sadly ill-nourished or rickety and hence poorly prepared to withstand the measles or pertussis from which they suffered at the time of, or shortly before, their admission to the hospital. It is therefore rachitis and malnutrition which Carr regards as the predisposing factors in the development of bronchiectasis when such children contract a specific bronchitis or broncho-pneumonia.

One child was described as badly nourished and pigeon-breasted, while another was fat and rickety. Still another was said to have been treated for congenital syphilis before admission to the hospital.

They all but one presented signs and symptoms of chronic bronchitis, generally of both lungs, with more or less extensive dullness at the base of one half of the thorax. The physical signs were fine or coarse crackling, or gurgling râles with bronchial breath-sounds which grew more distinctly tubular as the case progressed. The dullness usually remained as at first discovered, or, as in one case which developed in the hospital, became more pronounced with the progress of the disease. In one of the older children the entire left side was dull and retracted from an old-standing cirrhosis of pleuritic origin.

Difficulty of breathing was not so marked a feature as were cough and loss of strength and fever. In one instance cough was paroxysmal, but in none was there fœtor of the breath. "Sickness" is mentioned as occurring infrequently in a few cases. Pyrexia was occasionally high and irregular, but ranged on the whole between 99° F. and 101° F.

Debility was present in all whenever bronchitis was severe, but it was repeatedly noted that strength improved and the children ate well, although examination of the chest gave evidence of extension and increase of the bronchiectasis. Cyanosis was observed in some cases and in one clubbing of the fingers was mentioned.

The clinical picture was not distinctive of anything more than chronic bronchitis of the finer tubes and chronic broncho-pneumonia, and one can readily see why authors include bronchiectasis in young children under the head of these primary diseases. The course was chronic, generally lasting a number of months, and death was apparently from asthenia.

In every instance the autopsy disclosed an extensive system of bronchiectases which in more than one case were spoken of as reaching from the main bronchus to the periphery of the lobe involved. In the first child observed, the dilatations affected both upper lobes as well as the middle and right lower lobe. They were of the cylindrical variety and usually lined with smooth, sometimes congested, mucous membrane. Occasionally the dilatation involved the tubes throughout their extent even to the infundibula and resembled the fingers of a glove.

Such was the extent of peribronchitis that the intervening parenchyma was transformed into fibrous tissue, which in places formed but a thin sæptum between neighboring bronchiectases. In some instances there were evidences of chronic broncho-pneumonia, while in others there was only bronchitis. The tubes frequently contained thick pus or muco-pus.

Dense pleuritic adhesions of variable extent were often present, and in one the pleuræ and pericardium were uniformly adherent. In the sixth case it was evident from the density and toughness of the fibrous pleuritis that this inflammatory process had been responsible for the cirrhosis of the lung which had led to the bronchiectases. In one case the lungs were also infiltrated with small tubercles which were more or less cheesy.

Carr's cases are interesting and instructive from at least three points of view. (1) Considered with regard to the ætiology of bronchiectasis in children, they show that this is a very positive danger when measles or whooping-cough attack children suffering from rachitis or malnutrition in any other form. (2) From a pathological standpoint they prove that the evidence of changes of bronchitis and broncho-pneumonia may be entirely subordinated to the bronchial dilatation which they nevertheless induce. (3) Clinically they are of special interest since they illustrate the remarkable power possessed by young children to recuperate their strength, even in the presence of serious pulmonary disorders since some of these little patients actually improved in appetite and general health although the lung mischief was advancing. In this regard they accord with the statement made by Lenhartz that young children who give every appearance of glowing health may yet be sufferers from extensive bronchiectases. He made an autopsy on such a child who was killed by a fall from a window. The lungs disclosed bronchiectasis and yet the parents had not noticed cough in the child until two weeks prior to the fatal accident.

Physical Signs. **Inspection.**—There is nothing in the patient's appearance significant of bronchiectasis or indeed that may not be seen in other bronchial or pulmonary affections. In the earlier stages of the disease the appearance may, in fact, be that of perfect health, but as time goes on and the system suffers in consequence of complications or the wasting effect of profuse purulent expectoration the patient looks more or less cachectic.

In cases of long standing there may be cyanosis of the lips, clubbing of the fingers and other evidences of deranged circulation. The chest may be retracted on one side with compensatory spinal curvature and defective expansion of the entire lung or of only a portion, as apex or base. In other cases both lungs may show restricted respiratory movements, or the thorax may have the shape and rotundity seen in emphysema and known as barrel-shape.

Palpation.—In cases of chronic bronchitis and emphysema with bronchiectasis, palpation is of small aid, but when there is general or circumscribed fibrosis, tactile fremitus is increased over the whole or a portion of the affected lung. If the primary condition is an obliterative pleurisy, then fremitus is absent. If the bronchiectatic cavity is of considerable size and superficially situated, the hand may detect more pronounced vocal vibration over a circumscribed area than immediately round about, a circumstance that might be of value in strengthening other signs of cavity. Lastly, palpation may be of service in enabling one to determine displacement of the heart incident to retraction of a cirrhotic lung.

Percussion may or may not furnish information of great value according to the nature and location of the bronchiectasis. In associated bronchitis and emphysema the chest may be generally resonant or hyperresonant, the same as if the tubes were not dilated. In fibrosis or cases of old extensive pleuritic adhesions, there is dullness over the seat of the underlying pathological changes, or the note may be of a wooden quality owing to the combination of areas of induration and contiguous bronchial dilatation.

In favorable cases, percussion may elicit tympanitic resonance in a circumscribed area in the midst of dullness, a condition of things very suggestive of cavity. If such a combination is perceived in the upper lobe it may be due to tuberculous excavation, but perceived at the base behind or over the middle portion of the lung it is far more likely owing to dilatation of a bronchus.

A finding of great value but not pathognomonic, since it may occur in tuberculosis as well, is dullness in a circumscribed area at one time and a tympanitic note at another, e. g., directly after the expectoration of purulent secretion has emptied the tubes. This was brought out perfectly in the young man whose case has been cited several times already.

Such an abrupt change in the results of percussion following cough may be observed in phthisis, but in such a case it is more likely to be at an apex, whereas in bronchiectasis it is more often over the middle or lower lobe.

Care must be exercised by the inexperienced against mistaking for the alteration in the percussion-note just described the slight change from moderate dullness to normal resonance sometimes perceived at the base of a lung behind in chronic bronchitis after cough has cleared out the tubes.

Finally, if the bronchiectasis is superficial and its wall is sufficiently yielding it may give forth cracked-pot resonance on strong percussion. If it communicates with a bronchus of sufficient dimensions it may even yield Wintrich's change of pitch with open and closed mouth. Although the peculiarities in the percussion-note described are best elicited over a sacculated bronchiectasis of considerable size, still they may be obtained over an area filled with numerous, small dilatations which are separated by thin walls of fibrous tissue. Under such conditions the entire region has to all intents and purposes become converted into one huge cavity.

Auscultation.—The degree of information obtained by this means of examination depends upon the exact characters of the morbid anatomical changes and the state of the air-tubes as regards secretions at the time of auscultation. In emphysema with chronic bronchitis the chest may be filled with dry and moist râles which effectually hide all evidence of bronchial dilatation. Under favorable conditions, as in saccular bronchiectasis soon after expectoration, the breath- and voice-sounds may be highly suggestive of cavity, i. e., tubular or amphoric breathing, pectoriloquy and râles of a metallic quality. In the young man auscultation was negative so long as the bronchiectases were full, but immediately following expectoration the highly tympanitic area was filled with rhonchi of ever-varying pitch and timbre.

Diagnosis.—Under favorable conditions the recognition of bronchiectasis may be easy, while under others it may be extremely difficult. We possess no pathognomonic sign of bronchial dilatation, and yet the existence of chronic, perhaps paroxysmal cough resulting in purulent even fœtid expectoration, which gushes forth in mouthfuls, forms a feature so striking as strongly to suggest bronchiectasis. Should the effect of posture on cough and expectoration be well marked it strengthens greatly the conclusion. Nevertheless, even such a group of symptoms does not render diagnosis certain since, as I know by personal experience, precisely these characters may attend the periodic discharge of stinking pus from an empyema cavity that had ruptured into a bronchus.

The anamnesis must be studied with great minuteness, therefore, in any doubtful case. It was this which, in the instance just referred to, enabled me at length to determine that the condition was an empyema which had originated a few months previously from an unrecognized appendicitis.

The history of bronchiectasis is of a chronic affection which, as in the young man so many times alluded to, had existed for nineteen years and had its commencement in an acute broncho-pneumonia in his sixth year. The history, moreover, must include the fact of some affection either in the past or present which is capable of causing dilatation of the air-tubes as a part of its pathology. For instance there may be chronic bronchitis with emphysema or fibrosis of a lobe or of the entire lung.

In any case presenting such a history and with physical findings of some disease of the lungs bearing a close ætiological connection with bronchiectasis, the diagnosis becomes reasonably certain. If, in addition, there are the symptoms belonging to the clinical history of the complaint, reasonable doubt can scarcely exist even though percussion and auscultation do not enable one to locate the cavity.

Such an inferential diagnosis may suffice for the physician who does not contemplate operative treatment. It will not do, on the contrary, for the surgeon who is considering the possibility of pneumotomy or of producing collapse of the affected area. He must, if possible, have positive signs of the presence and of the exact seat of the bronchiectatic cavity. For the guidance of such it must be stated that diagnostic data on these points are unsatisfactory in very many cases.

For the surgeon it will not answer merely to be able to determine the existence of a cavity (the signs of which will be found in the chapter on the Diagnosis of Pulmonary Tuberculosis), since when he has ascertained the presence of a cavity he has in reality only entered upon the threshold of his difficulties. The two problems now confronting him are, (1) Do pleural adhesions exist? (2) What is the precise character of the bronchiectasis present?

As regards the existence or not of pleural adhesions we have no definite points of diagnosis. The history may assist in some instances. For example, the cirrhosis of the lung may have originated in or been subsequently associated with a pleurisy or a fibrinous pneumonia in which event the pleural surfaces are probably adherent. But if no help can be had from the anamnesis the question is to be answered, as far as possible, by the results of physical examination of the lungs, the play of the diaphragm, the retraction of the side and the position and immobility of the heart.

As concerns the kind of bronchiectasis present in any given case, on general principles it may be held that if well-marked signs of cavity are found in a case which, from the history and sputa, is bronchiectatic and not tuberculous, the dilatation is of such a nature as can be either collapsed or opened and drained. The case now to be narrated proves the insurmountable difficulties attending the differentiation of moniliform bronchiectases from a single cavity of considerable size.

Early in my practice I was called to treat a young woman with acute pneumonia of the middle lobe. In addition to the usual high temperature, rapid pulse, cough, scanty, bloody sputum and dyspnœa there were dullness, bronchial breathing and crepitant râles over a circumscribed area corresponding quite accurately to the middle lobe of the right lung. Crisis did not occur, and after a week or ten days the expectoration and breath assumed a very offensive odor while the appearance of the former strongly suggested gangrene. Unfortunately the sputa were not examined microscopically, but in the opinion of a colleague were unequivocally those of putrefaction of the pneumonic area. A few days subsequently I detected a small area of tympanitic resonance at the lower edge of the dull patch. Day by day this zone of tympany grew until it finally replaced the dullness, while the breath-sounds became intensely tubular in the same area and even at an opposite point on the back.

It was also noticed that whenever the patient coughed, this tympanitic area bulged out distinctly. Pectoriloquy was exquisitely marked and tactile fremitus was increased. It seemed beyond question, therefore, that a cavity had formed as a result of gangrene. By the time these changes had all taken place, the patient's condition began slowly to improve, the fœtor disappearing and the sputum gradually becoming purulent.

Convalescence was slow and after the woman had recovered, in a measure, I decided to have the late Dr. Christian Fenger see her with a view to surgical treatment of the supposed cavity by drainage. This was in the Fall of 1884, at a time when the journals contained much upon the surgical treatment of pulmonary cavities, and in my zeal I thought this cavity ought to be drained since it appeared so eminently suitable by reason of its superficial location.

Accordingly Dr. Fenger examined the patient carefully, corroborated my findings and concluded drainage was feasible. The woman consented to the operation and was removed to a hospital. I recall vividly that just prior to the administration of the anæsthetic Dr. Fenger went over the findings again and called attention to the bulging of the intercostal spaces caused by the act of coughing. It seemed as if there could be no doubt of the presence of a cavity of considerable size.

The surgeon used the actual cautery and experienced great difficulty in entering the cavity. At length, however, upon directing the cautery point outward and penetrating rather deeply he believed he succeeded in reaching the cavity and so expressed himself.

The patient did not do well after the operation, developed a severe pleuritis and in three days thereafter died. Only a partial autopsy could be secured, but this was sufficient to disclose the fact that no large pulmonary vomica existed.

Instead it was ascertained that the part of the lung in question was occupied by a series of small cylindrical bronchiectases which were arranged along the tubes precisely like a string of beads and constituted what are known as moniliform dilatations. What very small remains of pulmonary parenchyma there were in this area had been converted into fibrous tissue. To all intents and purposes therefore the multitude of bronchiectases had converted this portion of the right lung into a cavity, but one which by reason of its being subdivided by innumerable sæpta was inoperable.

Although this unfortunate case is most instructive from the standpoint of differential diagnosis, a matter that will be taken up a little farther on, it likewise teaches how deceptive may be a condition of multiple bronchiectases. One may be able to recognize the existence of bronchial dilatation, but not its exact nature as to shape, nor the number of cavities in every case.

In the present instance I should have suspected from the tubular rather than amphoric breath-sounds that a single cavity did not exist. The area over which tympany was present was so considerable that had there been a single vomica, there ought to have been other more definite signs. As a matter of fact it was the protrusion of the intercostal spaces which confirmed us in the opinion that a large comparatively superficial cavity existed.

This case teaches, furthermore, that an area of tympanitic resonance, pectoriloquy and bronchial breathing cannot be held to indicate a cavity, but that before the physician commits himself to a positive diagnosis he must carefully investigate all facts bearing on the case. Finally, more than a single examination may be required since the data obtained at one time may be quite different from those at another, especially if free expectoration has taken place just prior to the second examination. It may be added that an X-ray examination before and after expectoration ought to yield valuable information in locating a cavity.

Differential Diagnosis.—There are four conditions from which the affection now considered must be differentiated.

(1) *Putrid Bronchitis.*—This may not always be possible owing to the uncertainty of conclusive physical signs in bronchiectasis and to its associa-

tion with chronic bronchitis. In both affections the sputa may be putrid intermittently, but in fœtid bronchitis the expectoration does not occur so periodically nor in gushes, and it is not likely to have so offensive an odor as in bronchiectasis where the secretion remains for a longer time in the tube undergoing decomposition. Dittrich's plugs in the sediment would speak rather for fœtid bronchitis.

(2) *Tuberculous Cavity.*—(*a*) The history is likely to be that of a more rapidly wasting malady which began insidiously. (*b*) There may also be a history of tuberculosis in other members of the family. (*c*) Cough is more frequent and not paroxysmal. (*d*) Sputum is raised in smaller amounts and at frequent intervals and is not in sudden gushes nor likely to be of a fœtid odor. (*e*) The lesions are apt to be confined to or more pronounced at the apex of one and often of both lungs, this last circumstance being of much differential value. (*f*) Tubercle bacilli may be discovered in the expectoration and when so, speak strongly in favor of a tuberculous vomica, since tuberculosis secondary to bronchiectasis is uncommon.

(3) *Empyema.*—This affection is likely to be mistaken for bronchiectasis only when rupture into the bronchi has taken place and purulent sputum is periodically evacuated. In such a case the history is likely to furnish more valuable information than can physical exploration of the chest.

(*a*) The history of empyema is likely to be of a more recent affection and may be of an acute pneumonia with delayed convalescence, and then, after a longer or shorter period, of sharp pain in the side with sudden evacuation of a large amount of pus. In the case previously mentioned as resulting from appendicitis, the history was of an acute abdominal pain with gastric symptoms and fever, convalescence, and then, after a few weeks, an illness thought to be typhoid, again convalescence, and a few weeks later a pain in the right side with fever, cough and ultimately the sudden evacuation of stinking pus through the mouth. A subsequent operation substantiated the correctness of my diagnosis.

(*b*) It is not usual for an empyema discharging through the bronchi to give a history of such chronicity as does bronchiectasis, but if it should, the physical signs are not likely to be those of such well-marked fibrosis as is apt to exist in most instances of bronchial dilatation.

(4) *Pulmonary Abscess and Gangrene.*—A differential diagnosis between these affections and bronchiectasis may, under some circumstances, be most difficult. (*a*) In abscess and gangrene, the history is likely to be of an acute pneumonia during which occurred the very grave symptoms of lung necrosis with suppuration or putrefaction. (*b*) The symptoms of chronic abscess and gangrene are likely to be more severe than in bronchiectasis and rarely extend over so long a period. (*c*) The sputa are likely to contain hæmatoidin crystals, and in gangrene shreds of pulmonary tissue are more common, although the possibility of necrosis or putrefaction in bronchiectasis must never be ignored. (*d*) Cough and the manner in which expectoration is accomplished are not likely to be so explosive and peculiar as in bronchiectasis.

Prognosis.—It is stated by some authors that in exceptional instances the form of bronchiectasis which develops acutely as a result of acute bronchitis or broncho-pneumonia may undergo cure through retraction of the tubes after the subsidence of the primary affection.

The prognosis of the chronic variety is unfavorable from several standpoints. Unless the disease is amenable to surgical treatment it is incurable, and although it may pursue a very chronic course, the occurrence of some one of the many possible complications may at any time cause death. Some of the complications, e. g., cerebral abscess, are necessarily fatal, while others, as hæmoptysis and septicæmia secondary to decomposition of the bronchiectatic contents, although occasioning apprehension, are not necessarily fatal. Fœtor of the sputa is, therefore, a grave omen, since it may betoken the development of gangrene.

The prognosis also depends largely upon the nature and extent of associated or primary pathological lesions. Emphysema, in itself a serious malady, renders bronchiectasis a more grave affair than when the latter depends upon pleural adhesions or cirrhosis limited to a portion of one lung. A single not too extensive dilatation is more favorable than when a large system of bronchiectases exists, since it is more favorable for surgical treatment. When the disease has progressed to the development of cachexia, albuminuria, or circulatory embarrassment, the prognosis is most serious since the termination of life cannot be far distant.

Treatment. **Medicinal.**—In the majority of cases we can do nothing to bring about a cure of the disease and are consequently limited to remedies and measures likely to lessen symptoms. Of these, cough and expectoration are the chief so long as complications remain absent.

Cough may, according to Hoffmann, be almost incessant when a cavity is so situated in the upper part of the lung as to empty its contents into the lower portion through a nearly vertical bronchus leading from the dilatation. In such a case the indication would be, of course, to diminish the severity of the cough by heroin, codeine or even morphine. Ordinarily, however, this symptom is not so frequent as to require sedatives.

Expectoration should be furthered in all cases to prevent stagnation and decomposition of the secretion. Hoffmann recommends the prolonged use of expectorant remedies applicable to chronic bronchitis, and these have been discussed in the chapter dealing with that complaint. Fowler, on the other hand, strongly advises the employment of inhalations especially of the vapor of creosote after the manner recommended by Dr. Arnold Chaplin.

The method advised is as follows: A small room from which all draperies and furniture are removed with the exception of a wooden chair or two is selected for the "creosote vapor-baths." For the generation of the vapor ordinary commercial creosote is poured into a metal dish which is supported by a metal tripod, and underneath which a lighted alcohol lamp is placed. It is advised that the apparatus stand upon a flat stone or plate of sheet iron to catch the creosote as it runs over and thus prevent its soiling the floor. The doors and windows are now closed and the patient, sitting at a little distance from the alcohol lamp, inhales the creosote fumes as they fill the room.

The vapor at first excites severe coughing and hence the bath should not last for longer than fifteen or twenty minutes on alternate days. As the respiratory mucous membranes become accustomed to the fumes, the sitting may take place daily and be prolonged to an hour or more. Additional precautions recommended are that the clothing and hair be covered by cotton cloth, that the eyes be protected by close-fitting goggles, and that the nostrils be plugged with absorbent cotton.

Fowler's conclusions are, (1) It presents no practical difficulties and is unattended by any unfavorable results. The benefit to be obtained from the use of creosote vapor-baths in bronchiectasis is far greater than from any other form of treatment.

(2) In exceptional cases a condition almost amounting to cure may follow their prolonged use.

(3) As a rule the fœtor of the sputa is modified, and not infrequently the odor disappears altogether, but in some cases it is unaffected.

(4) The quantity of the sputa is not always or even as a rule diminished. It varies much from time to time and may be increased.

(5) The general condition of the patient improves, and those symptoms which remain cause less distress.

(6) The treatment requires to be continued for a long period, possibly as long as six months or more. In some cases its continuous use is advisable.

Intratracheal injections are also employed and have been recommended by Rosenberg, Granger Stewart, Colin Campbell and others. The remedies thus administered are for the most part menthol and guaiacol in olive oil, menthol 10 parts, guaiacol 2 parts and olive oil 88 parts, but other antiseptic agents may be used if in proper solutions. No irritation is produced by such injections if the nozzle of the syringe is carefully introduced through the chink of the glottis and vocal cords, so that the solution enters the trachea and not the larynx. This form of treatment may be given once a day. Excellent results have been reported.

Guaiacol and creosote in a 25-per-cent solution in sterilized olive oil have also been injected subcutaneously, the same as is sometimes done in cases of pulmonary tuberculosis. Ten c.c. are usually injected at a time into the loose cellular tissue of the chest between the scapulæ or in the axillary regions. In Germany good results are reported from the internal administration of myrtol up to 6 grams daily (Hoffmann). These last two methods are indirect means of disinfecting the bronchiectatic cavities and may consequently be considered less efficient than the creosote vapor-baths or intratracheal injections of suitable remedies.

Various tonics, as iron, arsenic, cod-liver oil and hypophosphites are also of service in improving general health and fortifying resistance and protecting against the wasting effect of daily copious expectoration.

Mechanical Measures.—Under this term Lenhartz highly recommends the method of forced expiration which was described in the treatment of chronic bronchitis. After taking a deep inspiration the patient aids expiration by bending his knees so as to make the abdominal muscles press upon the diaphragm, or at the same time he lowers his arms from the horizontal position to which they have been previously elevated, in such a manner as to squeeze the sides and front of his thorax. He is of the opinion that such powerful expiratory efforts exert positive influence in expelling the contents of bronchiectatic cavities, and therefore when the patient is too weak to execute the arm movements he advises compression of the chest by an attendant. If the lesion is situated only on one side, then that half of the thorax alone is subjected to the expiratory pressure. Lenhartz is positive he has seen improvement follow such a procedure. Its simplicity and its rationale certainly recommend it for trial. Any measure that tends to prevent stagnation and hence decomposition of the secretion within the dilated bronchus is worthy of adoption. Expiratory compression of the thoracic wall is certainly indicated in cases of bronchiectasis accompanying emphysema. It is doubtful if it would be of much value when there is pulmonary fibrosis with or without fibrous pleuritis. The thoracic wall is already retracted and the lung is indurated, so that it seems doubtful if any considerable degree of compression of the lung can be affected by the procedure described.

Surgical Treatment.—It is not in the scope of this work to enter into a discussion of lung surgery, even were I competent thereto, and it must suffice if only general considerations are stated. Medical writers and even surgeons appear not to be in harmony concerning the wisdom of surgical interference in this class of cases nor as to the precise technique to be employed. This divergence of opinion is owing largely to the difficulties in the way of determining the operability in any given case. By this, of course, is meant the exact location of the cavity or cavities, as well as their size and character. The question of surgical treatment must also depend upon the state of the patient's general health and the efficacy or inefficacy of medical treatment after the manner stated above.

Moreover, operative interference is not without danger, and hence it is not to be advised without serious consideration of the possibilities. Thus, of Lenhartz's 23 reported cases half the number died, as did 10 of the 39 operated on by Tuffier. Again, although the indications for surgical management may be present, it may be very difficult to decide just what sort of a procedure will secure the best results.

Godlee's recommendation to tap and drain, which is found in his and Fowler's work on "Diseases of the Chest," is considered very bad advice since there is no certain means of determining whether or not pleural adhesions exist, and when such are not present, leakage from the cavity into the pleural sac would be sure to follow. The indication is to secure drainage of the bronchiectasis by some perfectly safe means, and this may be accomplished either by producing collapse of the diseased portion of the lung or by incising and draining.

If it be decided to produce collapse of the lung and thus obliterate the cavity or cavities, then the bony portion of the parietes is to be removed and the case left for observation of the results. This simple procedure has been sufficient in some instances and yet it is quite possible for the benefit to be but temporary, when subsequent incision and drainage become necessary.

Should it be necessary to cut down upon the cavity and secure drainage, one should convince himself that pleural adhesions already exist or he should open the chest-wall without entering the pleural sac, and then set u irritation of the pleural membrane in order to occasion union of the surfaces. Only after this has been accomplished is the lung to be i the bronchiectasis to be drained.

Quincke is said to have first recommended resection of two or for the purpose of promoting collapse of the lung, and this is the indorsed by Lenhartz. One of the cases thus operated upon by th was that of a man who entered the hospital with signs of bronchiect left base behind and in a very bad general condition from septicæm the operation his general health improved steadily in consequence, a of obliteration of the cavity, since his expectoration became steadi amount and better in character. When at the end of five weeks h hospital his general condition was excellent. A year and a half l ever, his symptoms reasserted themselves and it became necessary and drain.

Climatotherapy.—In all cases which are not amenable to me surgical treatment a change of climate should be recommended. I be supposed that such a therapeutic measure is curative; it is only Prolonged residence in a mild, equable and dry climate tends, in a least, to diminish bronchial secretions and to invigorate the gener Consequently these patients generally improve when they live in which is favorable to out-of-door life and possesses the climatic stated.

CHAPTER VII

BRONCHIAL ASTHMA

NERVOUS ASTHMA

Definition.—The Greek term asthma signifies nothing more than dyspnœa or panting, but it has come to be restricted to a particular form of dyspnœa which is paroxysmal. The paroxysms or attacks occur suddenly and at irregular or periodic intervals, are characterized by extreme difficulty of both inspiration and expiration, chiefly the latter, by whistling râles, great distention of the chest and immobility of the diaphragm, and by the absence of fever or other signs of inflammation on the part of bronchi or lungs. The attacks usually subside as quickly as they set in and leave the individual perfectly well and free from shortness of breath.

Owing to the want of definite pathology and in consequence of the manner in which the paroxysms originate, the disease has received various appellations. Thus the form which arises independently of any recognizable organic disease in the lungs or elsewhere, is called *idiopathic, nervous* or *bronchial,* according as the writer chooses to emphasize what he conceives to be its pathological characters. On the other hand certain cases of paroxysmal dyspnœa associated with and probably depending for their causation upon disease of various organs have been recognized and are known by the following terms: *renal, cardiac, bronchitic, peptic, thymic* asthma.

These latter types, if such they may be called, should not be confounded with the pure nervous form, for the reason that they depend upon distinct morbid anatomical conditions, whereas the idiopathic does not, but in the minds of many is a pure neurosis. In this chapter it is the latter form that will be considered although the others will necessarily have to receive passing notice.

Pathogenesis.—This peculiar and distressing complaint has received much attention and chiefly for the reason that its pathogenesis and nature are not understood. The literature of the profession for the past hundred years or nearly so, teems with contributions to this subject and a number of different hypotheses have been advanced to account for the production of asthma. Even at the present day its exact pathogenesis cannot be said to be finally determined, and hence it is necessary to review the various hypotheses that have been promulgated.

Theories. Bronchial Spasm.—The theory most widely adopted and ably defended by Biermer is that of a tonic contraction of the circular muscle-fibers existing in the walls of bronchioles having a diameter of less than 1 mm.

or $\frac{1}{25}$ of an inch. In consequence of such spasm of the circular fibers the lumen of the bronchioles becomes diminished, or, in other words, the bronchioles become constricted.

This contraction of the small air-tubes interferes with both acts of respiration but chiefly the expiratory. Inspiration is more powerful than expiration since it can be assisted by powerful auxiliary muscles, and hence air can, with voluntary effort, be driven in past the point of bronchial narrowing. Since expiration is largely a passive act and can be assisted only in a limited degree by voluntary effort, it follows that the spasmodic narrowing of the bronchioles interferes with expiration more than it does with inspiration.

Breathing becomes laborious and slow and when closely scrutinized is seen to be peculiar. Inspiratory efforts are difficult and somewhat prolonged, but the expiratory are markedly prolonged and ineffectual. In consequence the thorax assumes a position of overdistention or, as it is termed, the position of forced and permanent inspiration.

Not only does the spasm of the bronchial muscles narrow the lumen of the tubes but these undergo still further compression by the greatly distended lobules and effective respiration is still further hampered. There now comes still a third factor into play, namely, spasmodic contraction of the great inspiratory muscle, the diaphragm.

Consequently, if the chest be now examined it is seen to be expanded to its utmost and to have assumed what is known as the barrel-shape of emphysema. Movements of respiration are nearly or quite abolished and percussion demonstrates distention of the lower lung border, as shown by depression and immobility of the upper line of hepatic dullness, hence motionlessness of the diaphragm.

At the same time the chest emits a multitude of high-pitched cooing or whistling râles which constantly change in position and intensity. It is this changeability of the rhonchi which furnishes the chief argument in support of the theory of bronchial spasm. Moreover, the possibility of such a spasm has been proven by animal experimentation by Donders, MacGillavry, L. Gerlach, Einthoven and Beer. The physiological proofs thus derived, when taken into consideration along with certain clinical facts, such as the extreme suddenness of both the onset and the abatement of the paroxysms, seem to its supporters to have placed the theory of bronchial spasm on a reliable basis. When, in addition, it is assumed that such spasm is excited by some influence acting on the nervous system in consequence of a neurosis, the theory seems to meet all the requirements of the case. Nevertheless this hypothesis is not received without reserve by many able men and hence another factor, namely, a mechanical one, is assumed independently of, or in conjunction with bronchial spasm. This additional theory will be mentioned later on.

Spasm of the Diaphragm.—Another theory advanced by Wintrich, and espoused by Bamberger and Lehmann, is that of a spasm or tonic cramp of the diaphragm. In common with all observers they recognized a tonic contraction of the inspiratory muscles, but maintained that this cannot be secondary to spastic narrowing of the circular bronchial muscle-fibers, since such a stenosis of the bronchioles would lead to elevation of the diaphragm, retraction of the thorax and sinking of the intercostal spaces. Instead of being diminished in its diameters the chest is forcibly expanded in the position of extreme and permanent inspiration.

Accordingly, Wintrich argued that as the diaphragm is the great muscle of inspiration, its contraction is necessary to expansion of the lungs, and as a matter of fact this muscle is contracted to its utmost limit during an asthmatic seizure. If the chest be percussed during a paroxysm it is found that the upper line of liver-dullness is depressed, thus showing that the diaphragm is in a state of contraction. Moreover, this line of dullness remains absolutely stationary, which proves that the excursion movements of this great inspiratory muscle are abolished. There is, therefore, a stillstand or spastic contraction of the diaphragm as the chief event in an attack of asthma.

The opponents to this theory of Wintrich claim that if such were really the case, there could be no expiration at all and hence they assert that Wintrich and his supporters have committed the error of mistaking the effect for the cause. This theory is now practically abandoned.

Fluxionary Hyperæmia.—A third theory suggested to explain the phenomena of spasmodic asthma is that of bronchial congestion. According to this hypothesis the dyspnœa is due to a mechanical impediment to the ingress and egress of air. This theory is not altogether new, but as a mere congestion of the bronchial mucosa cannot account for the suddenness of the advent and cessation of the paroxysm in cases of pure nervous asthma, an additional element had to be introduced.

Consequently Weber suggested an acute fluxionary hyperæmia of vasomotor origin as the true explanation of the attack. In support of his hypothesis Weber pointed to the instantaneous stoppage of the nasal passages that may be produced by acute congestion of the turbinate bodies, in response to mechanical or chemical irritation applied to the nasal mucous membrane, or indeed that sometimes occurs without any known cause.

That bronchial congestion is actually present during an asthmatic attack was asserted by Stoerck who claimed to have seen on laryngoscopic inspection redness and swelling of the mucosa of the trachea and large bronchi just beyond the bifurcation. It was pointed out, furthermore, that the cough and expectoration which occur at the termination of a paroxysm are additional proofs of the correctness of this theory. There also seems to be therapeutic confirmation of the correctness of this explanation in the oft-repeated observation that a cure of asthma may follow operative treatment of certain defects within the nares, as enlarged turbinates, which rendered the individual unduly sensitive to dust and other irritants. There is much to recommend Weber's theory.

Charcot-Leyden Crystals.—As remarked by Fraenkel, it was Leyden's special service to first point out the occurrence of certain rhomboidal crystals in the sputa of asthmatics. These bodies, which were subsequently proven to be identical with Charcot's crystals, possess sharp angles and edges, and hence were thought by Leyden to be capable of irritating the bronchial mucosa and thus of originating an attack of asthma. Their presence in the sputum is recognized by all, but that they are the direct exciting cause of this complaint is not believed.

The Recession of Skin Eruptions.—Another interesting and novel theory advocated by L. D. Bulkley in this country and the late Sir Andrew Clark in England is that asthmatic seizures are caused by local vaso-motor disturbances of the bronchial mucosa of nervous origin, the same as occur on the integument. It was based on the observation that patients suffering from urticaria sometimes have an attack of asthma directly following the disappearance of the nettle rash. This theory is allied to that of Weber but differs in some particulars. It is not generally accepted, and was opposed by D. R. Fraser on the ground that were Clark's explanation correct the inhalation of amyl nitrite would aggravate rather than relieve the attack.

Curschmann's Theory.—The discovery by Curschmann of certain peculiar masses of mucin in the expectoration of asthmatics led him to assume an inflammatory process, to which he gave the name of bronchiolitis exudativa as the essential cause of asthma. These so-called Curschmann's spirals are threads of mucin which present a twisted arrangement very like the twist of a corkscrew. The absence of all symptoms indicating inflammation renders this theory interesting but not tenable.

Of the various hypotheses just stated there are but two that need to be seriously considered. These are bronchial spasm and Weber's fluxionary turgescence of the bronchial mucosa. That the attacks are a manifestation of a disorder of the nervous system, a neurosis, seems placed beyond doubt, but precisely what takes place during a seizure is still questionable. There are so many facts going to bear out the correctness of both theories, namely, bronchial spasm and transient or coincident congestion, that I prefer the assumption of the association of both conditions.

There are some cases the peculiarities of which cannot be explained on any other hypothesis than that of a predominant if not independent narrowing of the bronchioles. On the other hand, there are cases which are more satisfactorily accounted for by the assumption of an acute inflammatory hyperæmia of the bronchial mucosa. It is very possible that in one case one factor predominates, while in another the other is the chief element. I cannot see why it is necessary to advocate one theory to the exclusion of any other. In the present state of our knowledge we can only say that bronchial asthma is a nervous affection which is probably excited by reflex action and consists of a combination of muscle cramp together with turgescence of the bronchial mucosa either inflammatory or of vaso-motor origin.

That a neurosis is a prominent if not the only factor in the pathogenesis of asthma is supported by several considerations on which Hyde-Salter lays particular stress. (1) Many of the exciting causes of an attack, e. g., mental emotion, fatigue, etc., are such as of themselves suggest the influence of the nervous system. (2) Most of the remedies effective in a paroxysm are such as act through the nervous system, namely, the nitrite compounds and antispasmodics. (3) A tendency is displayed by true nervous asthma to a periodical recurrence of the attacks. (4) Certain associated symptoms, as profuse limpid urine, neuralgias or frontal headache, unwonted excitability or hilarity, all point to its nervous origin. (5) The absence of discoverable organic change in the lungs of asthmatics also is a strong argument in favor of its nervous origin. All these will be brought out more forcibly in the following pages.

Ætiology. Heredity.—This appears to be a predisposing factor in many cases. Thus a child may develop asthma at the same age as did his father, or the complaint may befall several members of the same family in the same or successive generations. Hyde-Salter found what he believed to be a hereditary predisposition in 84 out of 207 cases. In some instances there may be no history of distinct inheritance of asthma but of marked neuroses in the parents, as hysteria, epilepsy, etc. Trousseau believed that such a neurosis in the parent might be followed by asthma in the child.

Locality.—The assumption of a nervous element in the causation of asthma is supported by the vagaries on the part of asthmatics as regards the influence of locality over the disease. Many instances are on record of the disappearance of paroxysms after change of residence, e. g. from Paris to Versailles (Trousseau) or from Paris to London where despite its fog and smoke one of Trousseau's patients enjoyed entire immunity for two years. The beneficial effect of change of locality is very apt, however, to be lost after the lapse of years.

The circumstance that asthmatics are very likely to again suffer from their old enemy upon returning to their former place of residence may be variously interpreted. It may be considered as merely the result of the impression made upon their unstable nervous system by the dread of a recurrence, or it may be argued that the locality is inimical because of the prevalence of some plant whose pollen excites the attack.

I once treated a lady who declared that no matter how bad her asthma might be she was always relieved so soon as she began a railway journey. The dust and smoke of travel did not affect her. In this instance the relief must have been achieved through the effect on her impressionable nervous organism rather than by the change of locality, for her relief came as soon as the train started.

Mental Impressions.—The influence of shock or imagination is shown in numerous cases. Thus Hyde-Salter relates the instance of a man who was seized by a violent paroxysm of asthma upon becoming alarmed by the sudden illness of his wife. One of Liebermeister's patients, cited by Lenhartz, invariably experienced an attack when traveling if he permitted himself to fancy the possibility of his being locked up in the compartment of the railway carriage in which he was at the time. In such cases of this nervous type, as it may be called, the mental impression manifests its effect not as an emotional excitement but as a paroxysm of dyspnœa resulting from a stimulus sent down the vagus to the lungs. Why the bronchi are the vulnerable point we know not any more than why embarrassment causes some persons to blush more violently than others or why apprehension excites a free flow of urine. Inasmuch as asthma has frequently been known to develop after an attack of measles, it may be that the bronchial tubes have suffered some unrecognizable injury to their histological structure which renders them abnormally sensitive. Such conjectures really explain nothing, however, and are only a begging of the question.

Reflex Irritation.—One occasionally sees the terms, *peptic asthma, uterine asthma,* etc., which are used to designate cases in which the exciting factor is supposed to originate in the digestive tract, or the pelvic organs, and to act in a reflex manner. A too hearty meal or one taken late at night, some indigestible article of food or constipation, have all been known to occasion an asthmatic attack, but the assumption that they act reflexly is open to doubt. It seems to me much more likely that in peptic asthma there is some toxin, a condition of auto-infection, which serves as an irritant to the nervous system and instead of producing a migraine or an intercostal neuralgia, as it does in many individuals, sets up in these neuropaths a paroxysm of asthma.

Some female sufferers from this complaint appear to have a special liability to their asthma at or about the time of their catamenia or during pregnancy. In such cases the explanation must be referred to disturbance of the nervous system which is so apt to attend this function, and with this assumption we must be content to let the matter rest for the present. I once treated a woman whose asthma often occurred during her menses and who also was especially prone to her attacks when the action of the bowel became more than ordinarily sluggish. At all events a course of colonic flushings which caused the disappearance of some old moth spots was also followed by an unusually long period of immunity from her asthma. The influence in her case of two such diverse factors supports the conclusion that an unstable nervous system was the ultimate cause of the asthma and nothing more. What there was in her constipation or in her menstruation to excite or disturb her nervous system, or why this disturbance was manifested by asthma, escapes our ken.

An instance of a so-called reflex cause is that narrated by Hyde-Salter of a man who invariably suffered from an attack whenever he got a wetting of the feet. This is akin to those cases of pseudo-angina pectoris in which an attack of pain follows the putting of the hands in cold water. I likewise know of a lady who invariably experiences pain and a peculiar clutching sensation in her right infraclavicular region near the shoulder whenever she places her feet in cold water. This lady once had a pneumonic abscess of the right lung close to the situation in which the pain is now felt as just stated. It is reasonable to assume therefore that the damage to her lung has rendered it sensitive to sudden changes of blood-pressure and that the irritation thus produced in the lung results in an impression upon the nerve centers. This is responded to by a sensation of pain in the intercostal nerves through an impulse passed down the spinal cord to the segment from which the respective intercostal nerves are given forth. By analogy one may assume that in cases of asthma there is also some condition within the bronchi which has impaired their integrity and makes them respond to impressions of various kinds which, in other people, are unattended by appreciable disturbance.

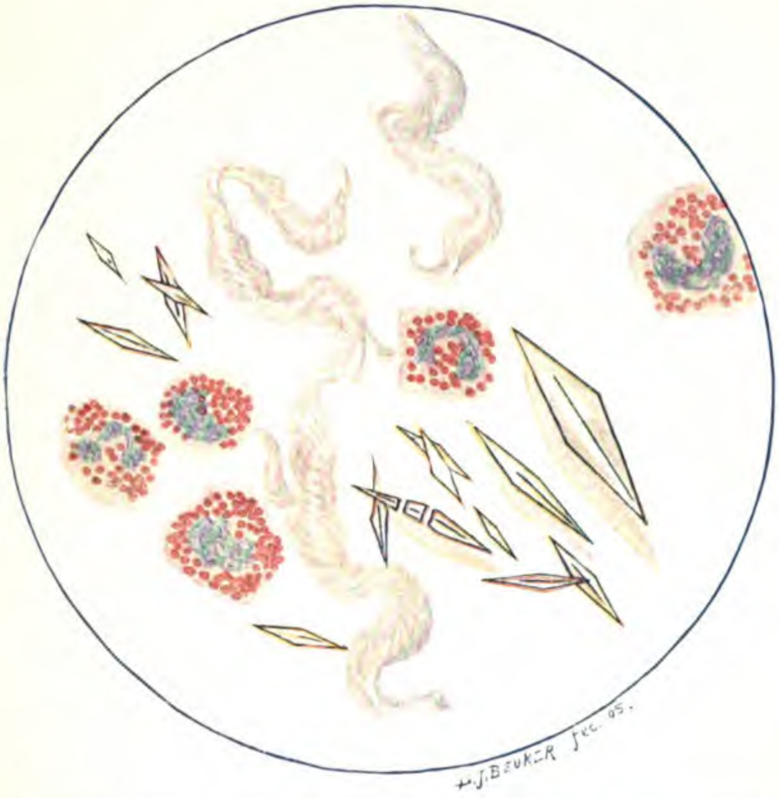
Nasal Diseases.—Another set of causes which may be included under the head of reflex is found in a number of intranasal disorders capable of exciting local irritation. The earliest of these to be definitely recognized and treated as a cause of asthma were nasal polypi to which attention was called by Voltolini in 1872. His observations were confirmed by other clinicians in Germany and elsewhere who added various other nasal defects to the list of such causes, such as deviations and exostoses of the sæptum, and chronic rhinitis. Hack, in 1882, laid particular stress on turgescence of the inferior turbinated body as a necessary concomitant of asthma, maintaining that the latter was a reflex neurosis analogous to migraine and other nervous affections. It has been proved, however, that Hack was mistaken in limiting the sensitive area to the anterior portion of the inferior spongy body, since irritation at other points within the nares is sometimes capable of exciting asthma.

Interesting and important as are pathological conditions within the nose in their relation to the complaint now being considered, it is even more necessary to remember that such a causal connection does not always exist. Indeed, the statistics of Lublinski, Schmiegelow, Herring and many others show that such an ætiological relation exists only in a small percentage of cases. From the standpoint of therapy also it is important to bear in mind that all observers are in accord in the experience that when asthma and some morbid nasal condition coexist, the former is not always relieved by removal of the latter.

Vegetable and Animal Odors.—In this connection it is appropriate to consider the influence exerted by odors given off by certain plants and by animals. There is a type of this disease known as hay asthma or hay fever which abounds in the Summer and Fall months, and undoubtedly depends for its causation upon the pollen of certain plants. It differs from ordinary bronchial asthma in the intensity of the coryza, with associated redness and watering of the eyes and in some cases more or less febrile temperature. There is generally also some degree of dyspnœa and hence its appellation, hay asthma. This is a misnomer, however, since it does not by any means always depend upon new mown grass and the asthmatic feature is subordinate to the coryza. In the United States it seems most often connected with that beautiful Autumn flower, the golden-rod. A feature of the disease that is quite distinctive is the almost unfailing certainty of occurrence on the same date each year and the regularity of its disappearance after a certain number of weeks, generally after severe frosts have killed off the flower on which it depends for its causation. Furthermore, immunity from it is enjoyed when its victims remove to localities free from the obnoxious flower.

There may or may not be recognizable lesions within the nose during the free intervals, but during the attack the cavernous tissue covering the turbinated bones is swollen and sensitive. It seems likely, therefore, that the disease is due to the local irritation caused by the pollen of the offending plant. The uncertainty of treatment addressed to the nose and the fact that not all individuals are affected in this manner point to a neurosis as the fundamental factor in its ætiology. This hay asthma cannot be regarded as identical with, but only allied to, the bronchial asthma we are now considering.

PLATE II



Asthmatic Sputum, Showing Eosinophile Cells, Charcot-Leyden Crystals, and Curschmann's Spirals.

On the other hand, it is interesting to note that paroxysms of true nervous asthma are evoked in some individuals by the smell or perhaps it should be said the dust of powdered ipecacuanha and by the odor of certain animals, as cats, horses, geese, etc. The sensitiveness of some asthmatics to goose feathers is so pronounced as to make it impossible for them to sleep upon pillows filled with them. I once knew a chronic asthmatic who used to remark facetiously that he never saw a goose in the street without wanting to run out and give it a kick. To the healthy individual free from idiosyncrasy of this kind such a hypersensitiveness of the Schneiderian membrane seems incredible except on the hypothesis of a neurosis, a supposition which is borne out by the great diversity of factors assigned for the causation of asthma.

Charcot-Leyden Crystals.—As already stated, the discovery by von Leyden of microscopic colorless octohedral or rhomboidal crystals in the sputa of asthmatic patients led him to advance the theory that the asthmatic seizures were due to irritation of the delicate bronchial mucosa by the sharp points of these tiny bodies (Plate II). This view is now known to be incorrect, since these crystals have been proved identical with those previously described by Charcot, which are found in a variety of other conditions. They are derived from spermine and consist of phosphate of spermine and are especially abundant in the blood, spleen and other organs of leukæmic patients. They occur also in anæmia and are not infrequently seen in the sputum of emphysema, chronic and even acute bronchitis. These crystals have consequently no significance either in the ætiology or diagnosis of asthma. From Hyde-Salter's description of certain peculiar bodies, seen by him in asthmatic sputum, it would seem that he perceived these crystals but without understanding their nature.

Age seems to exert only an indirect influence in the production of this disease, since it may occur at any time of life from infancy to advanced age. The following table is given by Hyde-Salter to show the ages at which the first attack developed:

During 1st year	11 cases	= 31.6 per cent
From 1 to 10 years	60 "	
" 10 " 20 "	30 "	13.3 "
" 20 " 30 "	39 "	17.3 "
" 30 " 40 "	44 "	19.5 "
" 40 " 50 "	24 "	10.7 "
" 50 " 60 "	12 "	5.3 "
" 60 " 70 "	4 "	1.8 "
" 70 " 80 "	1 case	0.4 "
Total	225	

Hyde-Salter met with instances of true nervous asthma in four infants of nine, fourteen and twenty-eight days and three months respectively. His observation has been confirmed by Politzer who saw it in two children of fifteen and sixteen months (West).

It is very common for old asthmatics to assert that they have suffered from their complaint all their lives, and as a matter of fact true bronchial asthma most often develops early rather than late in life. Indeed, whenever asthma originates after the fortieth year it may generally be found to depend upon some of the organic diseases soon to be mentioned. It may then be a bronchial asthma, but is, in its ætiology at least, different from that type of the affection which having begun in youth without apparent cause may be strictly termed nervous asthma.

When the complaint develops in childhood it may be traced to no particular antecedent disease, in many instances being thought to be inherited, but it not infrequently declares itself after an attack of measles or pertussis. It is this fact which has led some writers to assume some minute structural alteration of the bronchi as the predisposing factor, a supposition that seems warranted by the association of asthma with emphysema and bronchitis late in life. Nevertheless, not all children who pass through measles or whooping-cough develop asthma, and hence, as has been said so often in these pages, this fact warrants the conclusion that a neurosis underlies any possible pathological change in the bronchi.

Organic Diseases.—It is not at all uncommon for persons, more often men than women as it would seem, who suffer from chronic bronchitis, especially when associated with emphysema, to develop attacks of dyspnœa which so closely resemble idiopathic nervous asthma as to merit the term asthmatic. It is this form of asthma which it has most frequently been my lot to encounter. True bronchial or nervous asthma is relatively not a very common disease and as such is more often observed by the family physician than by the consultant. The latter sees it after it has existed for many years and has led to changes in heart and lungs, and in such cases I have seen the malady a goodly number of times.

When the asthma develops in consequence of chronic bronchitis and emphysema it is called *bronchitic asthma*, and in such instances it is not always easy to determine which antedated the other, the bronchial catarrh or the asthma.

Other types of the disease now considered are the so-called *renal* and *cardiac asthma*. The former is associated with chronic nephritis and, although it may display the characters of a true asthma, it is more apt to be of the nature of a paroxysmal exacerbation of an already existing dyspnœa. It is thought to depend upon the retention in the system of some of the chemical poisons which the damaged kidneys are not able to excrete.

Cardiac asthma, as its name implies, is in reality a paroxysmal dyspnœa symptomatic of cardiac incompetence from various causes, most often myocardial degeneration. It may occur as a result of physical effort but is most often nocturnal in onset, the attacks frequently coming during the hours immediately following midnight. The patient is very apt to be aroused from his first sleep by a sense of dyspnœa which causes him to sit up and gasp for breath. The similarity of this dyspnœa to true bronchial asthma is enhanced by the occurrence in the chest of wheezing and crackling râles. Very often there are cough and the expectoration of frothy or blood-tinged sputum. In such an event the attack is really one of acute pulmonary œdema and not asthma.

When the attack is one of paroxysmal exacerbation of an already existing dyspnœa and not of œdema, its features are very like those of spasmodic asthma and at the time may be differentiated with some difficulty. It is due not to bronchial spasm but to temporary increase of cardiac weakness.

Symptoms.—As asthma is essentially a recurring spasmodic dyspnœa we have to consider especially the phenomena of the paroxysm although it will also be in order to speak of the individual's condition during the intervals.

Mode of Onset.—This differs widely in different cases. In some the attack begins suddenly and without warning, the sufferer being plunged in a few minutes from a condition of easy tranquil breathing into one of most violent and even alarming dyspnœa. More commonly the "fit" is announced by certain sensations which, the victim knows by experience, surely presage his asthma.

Prodromata.—According to Hyde-Salter whose great experience entitles him to speak with authority, the most usual prodrome is an overpowering *desire for sleep.* This sleepiness is so extreme that although he knows his enemy will surely take advantage of his unconsciousness in sleep to attack him and that he can only ward him off by keeping awake, still the asthmatic, after vainly struggling with his drowsiness, at length yields to his irresistible desire and betakes himself to repose.

In other cases, on the contrary, the precursory symptom is a feeling of unwonted mental *excitation* or *hilarity* quite the opposite of the stupid, drowsy state just described. One is reminded by Salter's description of this mental excitement of the condition mentioned by Haig and by him attributed to a state in which the uric acid no longer circulates in the blood but is deposited in the tissues. I do not wish to be understood as advocating Haig's theory or implying that any such actual state as regards uric acid exists during this prodromal excitation of asthma. Such a condition of mental excitation seems sometimes to precede other distressing outbreaks, e. g., a migraine. It is as if the irritant were gathering its forces for an explosion, as a calm precedes a thunderstorm.

In some cases the premonitory symptoms are connected with the digestive apparatus and are of a nature which suggests, as stated by Salter, that they have a still closer relationship to asthma and are in reality its exciting cause. These symptoms are *flatulence* and *constipation* or other indications of indigestion. *Loss of appetite,* when it precedes an attack, may be regarded as more accurately a precursor than a determining factor. A very interesting and peculiar prodrome observed by Hyde-Salter in a single case was "ophthalmia." This conjunctivitis was not associated with coryza and was not therefore a part of catarrhal inflammation of the upper respiratory tract as in hay fever, but existed alone and was an unfailing indication of a forthcoming asthmatic seizure.

Another phenomenon which often attends the beginning of an attack, yet is not strictly premonitory, is *profuse diuresis,* the urine being nearly colorless like that attending hysteria and other nervous states. It is apparently of a purely nervous origin and not the result of toxic stimulation of the secreting structures. Neuralgic pains are also experienced by some asthmatics early in their attacks and indicate disturbance of the nervous system, in these instances I am inclined to think by some toxic product to which, perchance, the asthma itself may be due.

Features of the Attack.—In the majority of instances the paroxysm develops during the night, sometimes abruptly but more often by degrees. The patient retires either in his accustomed good health or aware through his usual premonitory feelings that his asthma is impending. Some hours after midnight, generally about four or five o'clock, he is aroused by a sense of difficult breathing and, sitting up, he endeavors to throw it off. If the wheezing is not yet severe, sleep may again overcome him; he sinks back into an uneasy slumber from which, after a brief interval, he is again awakened. In other cases the invasion is more sudden and sleep is permanently banished. In whichever manner the asthma appears it at length becomes so severe that the sufferer sits up in bed or, springing out of bed, makes his way to the window which he opens that he may obtain more air.

Most asthmatics now assume some favorite posture which experience has taught them best enables their auxiliary muscles of respiration to assist in the struggle for breath. This may be sitting, with elbows resting on the knees, or the arms supported on the back of a chair or a table, or standing, with the arms resting on a mantelshelf or high bureau. Salter states he has known an asthmatic to stand thus for thirty-six hours in the vain endeavor to minimize his sufferings. Whatever may be the position, it is one which enables the patient to fix his shoulders for the most effective action of his muscles.

The picture now presented is very characteristic and indicates a dire struggle against threatening asphyxia. The face is pale and anxious or, if the asthma has lasted for some hours, is of a livid hue. The veins of the forehead stand forth like blue tortuous cords and perspiration bedews the skin or trickles down in tiny streams. The eyes bulge from their sockets and are deeply congested while the head has the appearance of being sunken deeply between the elevated shoulders. The sterno-cleido-mastoid and scaleni muscles are contracted to their utmost and consequently stand forth prominently like huge immovable cords. The head is therefore fixed in its position turning neither to right nor left. The powerful muscles of the back, the trapezii and rhomboidii, are also contracted to aid in the attempt at respiration and consequently the back assumes a stooping or arched position which causes the sufferer to crouch or stoop over, like an old, feeble man.

While the eye of the observer thus beholds unmistakable evidence of intensest suffering the ear perceives a multitude of whistling wheezing sounds that proceed from the patient's mouth and are audible throughout the room. If the asthmatic attempts to talk, it is in single words uttered feebly and with difficulty as if he could not spare breath enough to articulate, while his voice sounds wheezy and muffled. Cough he cannot, and indeed in the height of his distress he has no desire to cough. His one thought and his sole desire is for air, without which, if relief does not come soon, he feels he must speedily succumb.

If at this time the chest is uncovered and inspected it is seen to be distended and motionless or nearly so, while the epigastric region is prominent and likewise lacking in its usual rise and fall as the diaphragm alternately descends and ascends with each respiratory act.

If the character of the respiration is studied at this time it is seen in most cases to be slow and laborious. The number of respirations have been known to sink to eight or nine in the minute. Exceptionally the respiratory rate may be accelerated (Salter), but marked slowing is the rule. This retardation is at the expense of both acts but chiefly of expiration. With inspiration every possible effort is made to force air in but such is the distention of the chest that further expansion is not possible and consequently the act is prolonged and futile. The air-vesicles are already filled to their utmost and consequently no matter how hard the patient tries still further to inflate his lungs in response to his need for air, he finds it utterly impossible to do so.

Ineffectual as is inspiration, expiration is still more difficult in consequence of the feebler force of this act. It is accordingly long and slow, so that it may be even four or five times the duration of the inspiratory act. It is this striking prolongation of the expiratory portion which gives to asthmatic breathing its peculiar and distinguishing type. Moreover, as pointed out by Salter, the pause that usually succeeds expiration is abolished and inspiration begins again as soon as the former act is completed.

In brief then, asthma is characterized by slow, laborious and yet ineffectual attempts to expand the lungs and by still more prolonged and equally fruitless efforts at expiratory emptying of them, while at the same time the chest is filled with singularly musical sounds known as sibilant râles. In consequence, therefore, of the mechanical distention that has taken place the chest remains apparently motionless despite the sufferer's unmistakable dyspnœa and struggles to overcome his threatening asphyxia.

If percussion of the thorax is made at this time the note is found to be everywhere hyperresonant, i. e., drumlike in its intensity and lowness of pitch. Præcordial dullness is obliterated and both breath-sounds and heart-tones are obscured by the abundant musical râles. The pulse is small, usually tense and slow in the beginning of the attack but feebler and more rapid after the asthma has persisted for a number of hours. The body temperature is not elevated but inclines rather to be subnormal.

Duration of the Attack.—Asthma is usually spoken of as occurring in attacks or paroxysms of variable length, and in typical instances of nervous asthma such is the case. The seizure may endure for a few hours or may drag on for a number of days with fluctuations in its intensity. Were it not for such brief periods of respite from his sufferings it is doubtful if the asthmatic could survive. When at length the dyspnœa finally ceases the individual enjoys an uncertain interval of entire freedom. There are cases however in which the asthma shows such transient and incomplete abatement that the sufferer may declare he never knows what it is to be wholly free from his complaint.

The following case may be regarded as a good example of this type of asthma in which the dyspnœa is so nearly continuous that distinct paroxysms of brief duration cannot be said to exist. It proves that periodicity does not by any means characterize all cases and that bronchial spasm may play a subordinate rôle to that of the hyperæmia of the mucous membrane. It is moreover a fair type of bronchial asthma as presented in an individual who had been free from the complaint until middle age and therefore can scarcely be said to present the idiopathic form of the malady.

Mrs. M. states that about five years ago she suffered from a severe attack of the *grippe*. She recovered from the worst of the attack but continued to cough during the remainder of the Winter. She was, moreover, very hard-worked and this, together with the persistent cough, got her into a very reduced state of health. At length, in June, she began to suffer from asthma and from that time until the date of my examination in June, 1905, she has been a victim of the complaint. She asserts positively that with one exception she has not been free from her trouble for more than a few days at a time. She has spent the past few months in the South and while there was so bad that her heart became weak, and swelling of the ankles developed.

At my examination she was found sitting on the side of the bed, in great dyspnœa. The air of the room was filled with the fumes of an asthma powder which she had been burning and which afforded her temporary relief. The face and cervical regions were suffused and puffy without being œdematous. The external jugulars were turgid and a pulsation was perceptible in the right vein. The ankles pitted slightly and the abdomen was distended apparently in consequence of congestion of the liver which was palpable and tender to pressure. The pulse was about 100, somewhat irregular in force and volume, and moderately tense. Her temperature was between 99° F. and 100° F. The lungs were hyperresonant; præcordial dullness was obliterated and the lower margin of pulmonary resonance was too low, but not entirely stationary. The chest emitted a multitude of sibilant râles on inspiration and a lower pitched groaning râle on expiration which was half as long again as inspiration (3 to 2). These râles were stationary, not shifting their situation as is described by Salter in cases of spasmodic narrowing of the tubes. The heart-tones were very feeble, and in the tricuspid area was a faint, soft, systolic murmur. The end of a lead pencil drawn across the skin of the chest produced a pale line which almost immediately resumed the natural color of the skin. Her expectoration was scanty and somewhat yellowish and only raised after prolonged and severe cough which she declared was frequent. She had slept but very little for several nights.

The condition was believed to be one of chronic bronchitis with asthma and she was given large doses of terpin hydrate with apomorphine, syrup of hydriodic acid, a brisk cathartic and tincture of strophanthus. A specimen of sputum was obtained and on examination was found to contain eosinophile cells, Charcot-Leyden crystals and one or two Curschmann's spirals.

The woman's condition slowly and gradually improved; the heart became stronger and after ten days or two weeks she was practically free from asthma. Nevertheless, she was still inclined to cough a little, although no râles could be heard. After a respite of three or four weeks she began to experience a "running from the nose" which she knew was the precursor of another asthmatic attack. In spite of a continuance of her expectorants and of the use of suprarenal extract dissolved on the tongue (see Treatment) she at length became so asthmatic that morphine had to be administered hypodermically at night. This attack persisted for another week or so and then gradually subsided under the use of increased doses of the former expectorants.

The last of July she went into the country and while there enjoyed five weeks of entire freedom from her asthma. She also gained strength and in fact felt better than at any time for the five years preceding. She was beginning to congratulate herself on having lost her asthma for good when a serous discharge from her nose again began, and after about a week was followed by a reassertion of her dyspnœa. This time the old enemy seemed to have gained new strength and when I again saw her, the first week in September, she was suffering extremely. As in previous attacks her cough was a very distressing feature and it seemed to her as if her head would surely burst.

Upon examination I found the chest filled with râles the same as at my first visit to her. Inspirations were accompanied by whistling, and the prolonged expiratory efforts by groaning sounds; and when, after a period of freedom from cough, the rhonchi became less marked a fit of coughing would bring them out again in greater number and intensity than before. This time, however, her temperature was normal and there was not such marked evidence of cardiac embarrassment. Blood-pressure registered by the Janeway apparatus was 150 mm.

A small amount of viscid sputum sent down to my office two days later was found to contain the same ingredients as before, but the eosinophile cells were not very numerous. There was a single typical spiral.

In this case the nasal symptoms which always usher in an attack seem to point to a reflex hyperæmia of the bronchial mucous membrane as the real pathogenesis of the asthma. Her nose has been cauterized for enlarged turbinates, she says, and for a time she thinks her asthma was somewhat better. Recent treatments by a specialist have not benefited her, however.

The *abatement of an attack* is generally gradual and yet, under some circumstances, e. g., the influence of strong mental excitement or emotion, the asthma may cease abruptly. Salter narrates the instance of a gentleman who, unable to move or speak, was so frightened by a sudden hysterical fit on the part of his sister that he jumped up, ran downstairs after restoratives and then up again to where his sister lay and was for the nonce wholly free from his asthma. An hour subsequently, after the strong mental excitement had passed, his asthma was upon him, as bad as before.

This case is interesting not alone because of the effect exerted by powerful emotion but because the mention of hysteria in his sister lends support to the theory of a neurosis being at the bottom of asthma. The sister was a neuropath as shown by her hysteria and in the brother the asthma was the form which his neuropathy took.

As has been already stated *cough* is absent during the height of the paroxysm, but as the attack begins to abate cough makes it appearance. In fact the appearance of this symptom is often the first sign of the approaching end of the asthma. The cough, at first difficult and not very productive, soon begins to result in the expulsion of a characteristic sputum.

Expectoration.—This is viscid, of a grayish white color and variable in amount. In most cases, especially in those of short duration, it is scanty; but when the asthma has persisted with varying intensity for days or weeks it may be quite profuse, and hence the distinction sometimes made of *dry* and *moist* asthma. The sputum is composed chiefly of mucin, covered by frothy serum and it is this richness in mucin which renders asthmatic sputa so viscid.

The striking characteristics of asthmatic sputa are not, however, either its tenacity, its color or its amount, but certain elements which it contains, viz., Curschmann's spirals, eosinophile cells and Leyden's crystals.

Curschmann's spirals appear to the unaided eye as minute, firm, sago-like pellets of a sticky consistency, but examined microscopically they are seen to consist of eosinophile cells and a central thread about which is twisted a mass of mucin, so that the central core as well as the envelope displays a more or less distinctly spiral arrangement (Plate II). That this peculiar body is composed of mucin and not fibrin has, according to Hoffmann, been conclusively proven by Schmidt who, in a highly favorable and carefully investigated case, was able to trace this material into the very finest bronchioles which were not only filled by the mucin but still preserved their epithelial cells intact.

It would thus appear settled beyond question that these spirals originate in the ultimate ramifications of the bronchial tree and that their peculiar twisted arrangement is due to the rotary motion to which they are subjected in consequence of the anatomical arrangement of the bronchioles. Chemically this secretion is characterized by its tendency to the formation of the singular crystals soon to be described, but whether it is due to an inflammatory process as claimed by Curschmann or is a specific "asthma-catarrh" (Hoffmann) is not yet established.

These spirals do not always show up in a typical manner. As Hoffmann states, the central thread may be more or less perfect but without its usual investment, or there may be a hollow cylinder of mucin which at the end tapers off to a solid thread, and the plug may show fine threadlike projections from its sides.

To revert a little more in detail to the corkscrew-like appearance of these spirals Hoffmann expresses the opinion that the bronchioles have such an arrangement. He thinks that if the bronchioles were straight they would not adapt themselves readily to the dilatation of the lungs incident to inspiration, but that if they possessed a spiral arrangement they would be capable of elongation and would thus facilitate lung expansion. In support of his argumentation he cites a beautiful metal cast of the bronchi in the pathological museum at Leipzig in which such a spiral form of some of the larger tubes is shown. Given such a peculiar arrangement of the finest air-tubes and a highly viscid secretion within them, then it is easy to understand how the compression to which the lungs are subjected would force the mucin contents of the bronchioles forward and thus impart to it its striking twist so like the turns of a corkscrew. But extreme tenacity of the contents is also a prerequisite since any other less viscid substance would be broken up and not pressed into a distinct spiral.

Eosinophile cells are another ingredient of asthmatic sputum and are said to occur in great numbers in Curschmann's spirals (Plate II). It is also interesting to note that Lenhartz claims to have discovered these cells in greatly increased numbers in the blood during an attack of asthma and, in very severe cases, even during the intervals. Von Noorden has shown that these cells are more abundant in the blood soon after an attack than at a considerable time thereafter. The significance of these elements and their relationship to Leyden's crystals are questions that have not yet been settled and hence do not require consideration.

Charcot-Leyden crystals have been mentioned twice already (Plate II). Owing to their transparency they are not always readily recognizable, especially in perfectly fresh sputum and when not very numerous. They grow more abundant after the expectoration has been exposed to the air for a time and then may be easily seen. According to Lenhartz their consistency is such that if the cover-glass be pressed down firmly upon the sputum a grating sensation is perceived.

With the oncoming of cough and expectoration the asthma begins to abate, in some cases disappearing so rapidly that the patient feels entirely free from dyspnœa within a few hours. In other cases its disappearance is much more gradual and the individual is left much exhausted. In persons whose heart and lungs have not suffered the organic changes that eventually develop after many years of frequent and severe asthma the subsidence of the paroxysm is succeeded by an interval of seemingly good health.

Since beginning the writing of this chapter I have been consulted by a man of twenty-three who gives a history of asthma since his fifth year and who was, with the exception of occasional attacks of palpitation, in excellent physical condition. At the age of five he was ill with pneumonia and from that time on he was a great sufferer from asthma which kept him in delicate health up to his twelfth year. Since that age his asthma has been less frequent and less severe, coming on now when exposed to atmospheric and other conditions which, in most persons, are followed by symptoms designated as a cold. It is also induced by the inhalation of dust, and in this connection it may be stated that he acknowledged having always had nasal catarrh for which he is to receive treatment. Dietary indiscretions appear to exert no influence upon his asthma, for he is in the habit of taking a hearty meal of beefsteak with half a dozen cups of tea just before retiring for the night, and also of eating large amounts of confectionery daily. As a college student he took an active interest in athletics but since going into business has taken no other exercise than walking. In consequence of this and of his indulgence in confectionery he has increased rapidly in weight and is now on the verge of corpulence.

His asthma is not ushered in by prodromata but is attended with great nervousness and usually lasts two or three days. It subsides quite abruptly and during the succeeding period of freedom from dyspnœa he feels perfectly well. The attacks of palpitation for which he sought advice were not associated or in anywise connected with his asthma but developed some five years ago apparently in consequence of severe exertion one hot Summer while bathing in the surf off the New Jersey coast. Since that time the symptom has annoyed him at irregular intervals and apparently without special exciting cause unless it is the state of his digestive organs.

Physical examination was quite negative both as concerned lungs and heart. The chest was well developed and resonant throughout, but without a hint of emphysema. At the right base there were two or three faint sibilant râles on inspiration which might have been taken to indicate a slight catarrh of the smaller tubes, a reminder, as it were, of their liability to spasm. The heart was not demonstrably enlarged and its tones were quite clear. There was nothing to explain its occasionally disordered action.

Only two explanations seemed reasonable. The palpitation may have been owing to irritation by toxines of indigestion or the products of defective metabolism imperfectly eliminated, and considering his gastronomic indiscretions, this seemed not unlikely. Or it was an expression of that easily perturbed nervous system which ordinarily declared itself as an asthmatic attack and which for the last nine years had been of a milder type than formerly. On the whole, the case illustrated very well the condition, between attacks, of the asthmatic who has not yet suffered appreciable organic damage.

Quite different from the foregoing is the condition of the asthmatic who has been a victim of his complaint from early childhood to old age. In him both heart and lungs have become organically diseased and he bears about with him unmistakable signs of his malady. Even before he has grown bowed by the weight of years he presents the appearance of premature age. His face has an anxious, wrinkled look, lines of suffering being especially marked about the nose and mouth. His head is bent forward between his elevated and stooping shoulders while the back is permanently arched with the shoulder blades spread wide apart. The chest presents the barrel-shape of permanent emphysema being particularly full and round in its lower zone, owing to more or less obliteration of the angle formed by the junction of the costal cartilages and ribs. The upper portion of the abdomen also looks full because of the permanently low position of the diaphragm, while in the epigastrium may be seen and felt the pulsation of the hypertrophied right ventricle. The superficial veins stand out with unnatural distinctness and there may be some cyanosis of the lips. The chest gives forth a highly resonant percussion note and even when asthma is not actually present the breath-sounds are apt to be accompanied by rhonchi at the bases. The heart-sounds are indistinct or muffled.

In this stage of the disease chronic bronchial catarrh and emphysema have become established and consequently there is at all times more or less shortness of breath which readily displays a slight asthmatic quality whenever the patient hurries a little in his walk, encounters a strong wind or even indulges in a fit of laughter. He has a chronic cough of varying severity and with a rather scanty amount of viscid sputum. His voice is apt to be husky and often decidedly wheezy particularly when he laughs which excites an attack. In consequence of the circulatory disturbance which has been induced and also because in many cases digestion is defective, and this indigestion is apt to evoke a paroxysm, the patient finds his appetite impaired and takes but little food. His nutrition suffers, he grows thin, gradually loses strength and during inclement weather is obliged to remain indoors.

Thus the general health fails progressively although slowly until, at length, the old asthmatic becomes practically a confirmed invalid. He is an easy prey to attacks of acute bronchitis which greatly aggravate his chronic bronchial catarrh and at length he is confined permanently to the house or even to his apartment. In this pitiable plight his last weeks are passed in an easy chair in the condition of the sufferer from some other pulmonary complaint, e. g., from emphysema, or in that of the cardiopath with dropsical lower extremities, feeble, rapid heart, the chest filled with all sorts of râles, unable to lie down and scarcely able to speak for want of breath. The end comes either gradually with the phenomena thus briefly pictured, or a terminal pneumonia proves a merciful deliverer.

I recall a man in this distressing condition who had been an asthmatic only about a dozen years. There was a history of chancre and for this reason he was given large doses of potassium iodide which for a time gave such relief that we both began to think him about cured. After some months, however, the asthma gradually returned and at length put him in a worse condition than he had been before.

An interesting fact in this man's case was his positive statement that he was always worse at home and, on the other hand, comparatively free from dyspnœa as soon as he got back to work in the shop where he did cabinet work. There must have been more or less dust in the atmosphere of the shop and yet he experienced no discomfort; on the contrary, he was always glad to reach the shop after a night of suffering. This man ultimately died in very much the condition described above.

Physical Signs. Inspection.—The appearance of the asthmatic during his attack is one of extreme suffering. Even to the unaccustomed lay spectator it is evident that the person is struggling for air, but as this is also the case in several other conditions to be considered in the differential diagnosis, it is necessary to describe the peculiarity of the respiratory difficulty present in asthma. The posture and general appearance have been already portrayed under the heading of *features of the attack.*

Although the asthmatic is plainly on the verge of asphyxia it is not because of impediment to inspiration but on account of his inability to expel the air during expiration. His chest is therefore expanded to its utmost capacity and is nearly or quite immobile. Inspiration is short and jerky while expiration is abnormally prolonged and ineffectual, four or five times as long as inspiration. In most instances, therefore, the entire respiratory cycle is slow and labored so that the total number of respirations in a minute is reduced rather than increased. The distinguishing characteristic to be noted, therefore, is the difficulty of the expiratory in contrast to the inspiratory effort. This is readily seen but the emphysematous distention of the thorax can only be fully appreciated by removing the clothing and watching the limited play of the naked chest.

Palpation is of negative value since the degree of dyspnœa and the hyperexpansion of the lungs are best determined by the eye. Pectoral fremitus is decreased and the impulse of the heart is indistinguishable excepting, it may be, in the scrobiculus cordis, where, because of temporary cardioptosis produced by the descent of the diaphragm, the dilated right ventricle may be felt to pulsate strongly. The pulse may be either retarded in rate or accelerated and is deficient in volume. In my experience the pulse is apt to be slow and tense in the early part of an attack, but accelerated, less hard and often irregular after the paroxysm has lasted for two or three days and the heart begins to flag somewhat under its strain.

Percussion.—If the chest be percussed during a fit of asthma the note is everywhere hpyerresonant, the area of absolute cardiac dullness is obliterated and the inferior margins of the lungs are found to extend much lower than normal, reaching in some instances even to the level of the eighth or ninth costal cartilage in the vertical nipple line. Not only is proof thus obtained of the permanent descent of the diaphragm but the upper line of liver dullness which ordinarily descends and rises with each alternate respiratory act remains stationary. It was this absence of normal excursion movements on the part of the diaphragm which led Bamberger and others to conclude that asthma was due to spastic contraction of this great inspiratory muscle. It is now believed, on the contrary, to be a result and not the cause of inspiratory distention of the lungs. This immobility of the lung borders constitutes, therefore, a strong point in the differentiation of asthma from some other conditions also characterized by dyspnœa. It also furnishes incontrovertible proof that the difficulty of breathing in this affection is expiratory.

Auscultation.—It does not require the application of the ear or the stethoscope to the chest-wall to detect the peculiar sounds which issue from the lungs during a paroxysm of asthma. Even at a considerable distance, sometimes throughout the room, is heard a multitude of râles generally described by the patient and friends as wheezing. They are as a matter of fact sibilant with a whistling, cooing or squeaking character, and seem to proceed from all portions of the thorax so that the chest may not inaptly be likened to a music box made up of an infinite number of pipes which are played without harmony or purpose.

In a typical case of spasmodic asthma the adventitious sounds always present these characters, and during the acme of the paroxysm are never moist and crackling. Râles of the latter sort only appear as the attack abates when cough and expectoration come on, and even then they are apt to be mixed with whistling rhonchi.

Another peculiarity upon which Salter laid great stress, but which, it must be acknowledged, is not present in every case, is the instability of the râles. At one moment they are heard with special distinctness and in greatest profusion over some one area whence, at the next instant, they have disappeared in a measure to be perceived most loudly and abundantly in some other situation. It is this inconstancy which he advanced as one of the strongest arguments in favor of the spasm theory of asthma. As very pertinently pointed out by him, a catarrhal swelling of the bronchial mucous membrane could scarcely come and go with such rapidity as to cause the almost instantaneous shifting of sibilant râles to different parts of the lungs. Finally, such is the multitude of râles and such the feebleness of respiration that breath-sounds proper are inaudible.

Diagnosis.—There is usually little or no difficulty in determining the real nature of an asthmatic seizure. In the first place there is a history of recurring dyspnœa which is of intense type, is independent of effort, comes on more or less suddenly, lasts for hours or days, then departs as quickly, it may be, as it came and is followed by a period of complete immunity during which the individual can exercise or attend to business without any difficulty of breathing.

If the patient be seen for the first time during the height of his suffering and a definite history cannot be obtained a proper attention to the type of breathing and to the physical signs just described ought to render error of diagnosis impossible. The main clinical findings on which reliance must be placed are *the expiratory character of the dyspnœa and the sibilant nature of the râles.*

The real difficulty lies not so much in recognizing the nature of a genuine fit of bronchial asthma as in determining whether some attacks of dyspnœa called asthma by the sufferer are in reality asthma and not some other form of dyspnœa, perchance of a far graver import.

Differential Diagnosis.—The diseases which must be distinguished from asthma are the following:

(1) *Laryngeal stenosis* of organic origin, e. g., diphtheritic, syphilitic or cancerous. (*a*) There is no history of recurring dyspnœa but rather of gradually increasing difficulty of breathing which has at length culminated in extreme and persistent dyspnœa. (*b*) In syphilis there may be history of previous infection, in cancer, of a possible tumor in other situations, in membranous croup history of diphtheria in the family or neighborhood or of previous false membrane in the throat. (*c*) The difficulty of breathing is inspiratory as shown by stridor, extension of the neck and sinking of the soft parts, e. g., the cervical regions, intercostal spaces and epigastrium. (*d*) Laryngoscopic inspection is likely to detect the cause of the obstruction.

(2) *Spasm of the Glottis.*—(*a*) Spasmodic croup is most frequent in young children who have been previously well. (*b*) There may be a history of previous attacks occurring at night and subsiding spontaneously or after the administration of simple home remedies. (*c*) The dyspnœa is inspiratory with stridor and there is hoarse, croupy cough. (*d*) The attack generally subsides abruptly when the child seems in imminent danger of asphyxia.

(3) *Glottic spasm* due to irritation of the recurrent laryngeal nerve from pressure of an aortic aneurysm. (*a*) There is apt to be a history of previous attacks of short duration, from minutes to an hour. (*b*) It occurs most often in men with a specific history. (*c*) The dyspnœa is inspiratory as in other forms of laryngeal obstruction with stridor, etc. (*d*) There may be signs and

other symptoms of aneurysm. (*e*) Examination of the left lung may show signs of bronchial narrowing. (See Bronchial Stenosis.)

(4) *Œdema of the Glottis.*—(*a*) There is not likely to be a history of previous attacks but of dyspnœa which has set in gradually and has steadily, perhaps rapidly, increased to its present intensity. (*b*) The difficulty of respiration is inspiratory and is of the characters described in the foregoing conditions. (*c*) There are probably signs of cardiac or renal disease with dropsy elsewhere or of some mediastinal growth which is producing pressure. (*d*) Inspection of the throat reveals œdematous swelling of the epiglottis and surrounding tissues. (*e*) The dyspnœa is relieved by incision of the infiltrated tissues.

(5) *Aneurysm of the Aorta.*—This is probably the condition which most often has to be differentiated from asthma and is perhaps the most likely to lead to error. This is especially the case when the aneurysm is small and is therefore an aneurysm of symptoms and not of signs, as is said. Spasm of the larynx from aneurysmal irritation of the recurrent nerve has already been considered. In addition, asthma may also be simulated by compression of the air-passages below the larynx and when such is the case attention should be paid to the following points. (*a*) Dyspnœa is constant in some degree although it may be subject to exacerbations. (*b*) It is chiefly inspiratory as shown by stridor, and coarse mucous râles below the seat of constriction or in one or both lungs. (*c*) Careful study of the case will usually detect signs of the tumor on the part of the circulatory apparatus. (*d*) There is apt to be dullness either circumscribed along the course of the arch or of the lung subjected to pressure. (*e*) In some instances the dyspnœa becomes paroxysmal only when some certain posture is assumed as the dorsal decubitus. (*f*) It occurs chiefly in men who give a history of syphilis. (*g*) X-ray examination is likely to reveal the presence of the aneurysm.

(6) *Tracheal or Bronchial Stenosis.*—Aside from compression by aneurysm the air-tubes may be occluded by malignant growths and other conditions enumerated in the chapter devoted to that subject. The main differential points are (*a*) The constancy and inspiratory nature of the dyspnœa similar to that caused by a vascular tumor. (*b*) The history of some affection likely to lead to occlusion or compression of the air-tubes. (*c*) Physical signs indicating interference with the entrance of air into one lung or a portion of one lung, or, if the trachea is occluded, of collapse rather than emphysematous distention of both lungs. (*d*) Tracheoscopy may disclose the presence .(constriction) of a foreign body, etc.

(7) *Cardiac Dyspnœa.*—In a considerable number of cases this form of dyspnœa may so closely resemble spasmodic asthma as to render differential diagnosis difficult, particularly if the attack is not actually witnessed by the physician and he has to rely upon its description. (*a*) In its paroxysmal intensification of an already existing dyspnœa of effort the difficulty is not so much in the expulsion of air from the lungs as in the gratification of a veritable air-hunger which impels to a more hurried and deeper respiration than normal, thus evincing no actual impediment to either the ingress or egress of air. (*b*) The labored breathing is usually accompanied by moist râles or by these together with wheezing rhonchi. (*c*) There may be cough as well

as dyspnœa and there may be in some cases frothy expectoration. (*d*) There are usually objective signs of cardiac or cardio-vascular disease. (*e*) Attacks of cardiac like those of bronchial asthma occur most often at night but they are rarely longer than an hour or two in duration.

In most cases the discovery of some heart lesion renders the differential diagnosis easy, but now and then a case is encountered which is extremely puzzling. At the present writing I have under treatment such an instance.

The patient is a lady of forty who gives a history of having been a blue baby for a number of days after birth and now presents plain signs of cardiac disease which are not easy of interpretation. There is in addition to the ordinary findings of mitral stenosis a loud rasping murmur of greatest intensity over the base but not apparently belonging to any particular area. It is suggestive of patency of the *foramen ovale*, an inference that seems borne out by the great size of the right auricle. There are subjective symptoms of cardiac inadequacy.

This patient declares she never knew she had anything the matter with her heart until about six years ago. At that time she was in California where she spent several weeks in the mountains riding horseback and exercising in other ways without discomfort. She then went to San Francisco and on reaching there experienced an attack of dyspnœa which she called asthma. Inquiry elicited certain facts, however which seemed to indicate that the attack was more like one of acute pulmonary œdema than spasmodic asthma.

Nevertheless, whatever may have been the exact nature of that attack it left her with a distinct tendency to paroxysms of difficult breathing which have been regarded by physicians, as well as by the patient, as asthmatic and have prevented much exercise. In particular the patient has not been able to indulge in horseback riding at sea level; and yet a couple of years ago when in Asheville, North Carolina, at an altitude of 2,200 feet, she was able to ride without any respiratory difficulty. The past Winter, i. e., 1903, she had an especially severe time with her asthma for several weeks. Careful questioning convinced me that her dyspnœa was not typically asthmatic; for although it compelled her to sit up in bed and was most troublesome at night it was accompanied by coughing and sometimes by expectoration of frothy mucus. Moreover, it appeared to be physical effort that evoked the dyspnœa rather than anything else.

These considerations together with the discovery of a definite organic lesion led me to express the opinion that the asthma was not true bronchial asthma but was cardiac. The lady was accordingly advised to go to bed and submit to such other therapeutic measures as would be likely to restore some degree of cardiac competence. The advice was acted upon and for a time improvement was shown by complete disappearance of her asthma.

This change for the better was attributed to the milk diet and use of cathartics which not only lessened venous and hepatic stasis but reduced blood-pressure, as registered by Gaertner's tonometer. Things seemed to be progressing satisfactorily when, after the lapse of about two weeks, her breathing again began to be troublesome and just before the appearance of her menses grew so intense as to compel her to sit up in bed the greater part of each night.

My visits were always made during the daytime and generally in the forenoon, so that I never saw her in one of her asthmatic seizures and, as a matter of fact, really never observed any actual paroxysm of dyspnœa. Her respirations were accelerated but she was not asthmatic. Here then was an added element which I could not understand. Exertion occasioned difficulty of breathing which might reasonably be attributed to cardiac weakness, but in addition there was nocturnal dyspnœa which probably was connected with the catamenia since she stated she was apt to suffer from her asthma at this time in the month.

Her dyspnœa was always attended by cough though not always by expectoration, and in response to my inquiries she stated that her wheezing displayed rather the character of rattling than of whistling sounds. It should now be stated that upon giving her chest a careful examination a few days after her asthma had reasserted itself I was surprised to discover dullness and fine dry crackling râles with both acts, but chiefly with inspiration, at the right base from scapula to diaphragm. These findings had not been detected two weeks earlier at my office and hence were thought to indicate hypostatic congestion. She was not having any rise of temperature although she complained of a sensation of heat which made her tolerate very little clothing. It seemed plain, therefore, that the pulmonary condition was not inflammatory. Cough was a rather troublesome feature at this time and necessitated the use of heroin which relieved also the dyspnœa. It was not well borne otherwise, however, for when, on one occasion, she was compelled to take several of the heroin tablets each containing $\frac{1}{12}$ of a grain, she became very nauseated and vomited, being unable to keep anything on her stomach for nearly twenty-four hours. This experience was trying but it nevertheless had the salutary effect of removing her asthma while the nausea lasted.

Sips of hot water and minute doses of calomel and salol corrected the state of her stomach, after which it was decided to try the effect of large daily doses of terpin hydrate. Accordingly five grains were administered four or five times daily, and under its influence her symptoms gradually improved. The cough slowly disappeared, and the expectoration which developed after the administration of the terpin hydrate also gradually left. At the same time the physical findings at the right base gradually became less pronounced. The lung did not wholly clear up, however, and the râles finally displayed the characters of pleuritic friction. So that I came to the conclusion that there were old fibrous adhesions in this situation, although no history of a pleuritic attack could be obtained.

Prognosis.—In determining the prognosis of a given case of asthma several things are to be considered: (1) The likelihood of death during a paroxysm; (2) the prospect of cure either spontaneously or by treatment; (3) the influence of the asthma upon the health of the patient and upon his duration of life.

(1) Authors are generally agreed in the assertion that true nervous asthma does not destroy life *per se*. The presence of some complication as cardiac disease may render an asthmatic seizure dangerous and the persistence of a severe attack for many days or weeks in an old person whose lungs or heart

have become damaged by frequent repetitions through many years may imperil life through exhaustion. In the young and otherwise healthy, on the contrary, a fatal termination is not to be expected no matter how alarming the symptoms. Consequently, as a general proposition, it may be stated that the physician may have no hesitation in assuring both sufferer and friends of his recovery from a paroxysm. As a rule, no fear on this score is entertained by the patient who has learned from experience that relief is sure to come in time.

(2) The question most likely to be propounded to the physician relates to the prospect of the asthmatic's eventually obtaining permanent relief from his malady. On this point a guarded prognosis should always be given. There are certain considerations, however, which may be borne in mind as influencing prognosis in this regard.

(*a*) *Ætiology.*—If the cause appears to lie in some recognizable structural defect in the nasal passages, there is a possibility, though by no means certainty, of permanent relief from the asthma, following appropriate surgical treatment. Many brilliant results of this kind have been reported from all parts of the world, but on the other hand there have been more failures than successes. In verification of this statement I may be permitted to quote the figures given by West.

	No. of Cases.	Cured.	Improved	No Result.
Lublinski	143	27 (= 18.8 per cent.)	13 (= 9 per cent.)	....
Heymann	54	29 (= 55 ")	14 (= 27 ")	11 (= 18 per cent.)
Schmiegelow	50	32 (= 57 ")	11 (= 20 ")	7 (= 12 ")

If the asthma is of the purely nervous type and no other cause can be discovered than heredity, or if it seems due to some histological alteration of the bronchial passages occasioned by measles or other disease in early childhood, then the prospect of permanent cure is not encouraging. Cases of asthma which develop in association with or possibly as a result of chronic bronchitis and emphysema furnish an unfavorable prognosis, since they rest upon a pathological basis which itself is unfavorable as regards entire recovery.

(*b*) *Age.*—Salter is very positively of the opinion that the age of the patient has much to do with the prospect of cure. Children under ten are, according to him, quite likely to outgrow the complaint. When, on the contrary, asthma develops at or near the age of forty, or when the asthmatic has reached this age without relief, the prospect of cure is extremely doubtful. When, according to Salter, the complaint first appears after middle age it can probably never be cured because it rests upon some serious organic change in the heart or lungs. An additional reason for the unlikelihood of permanent cure late in life lies in the matter of habit. By this is meant that chronic asthmatics appear to have suffered some definite peculiarity of the nervous system, i. e., of the nerve centers, which may be said to be a fixed habit by virtue of which nerve irritation is responded to in the form of asthma, even when the original cause may have been removed. On the other hand it is the readiness with which children outgrow a habit which, in Salter's opinion, has much to do with their recovery from asthma.

(*c*) *Frequency and Duration of the Asthma.*—The more frequently the attacks of asthma occur, the less likely is the prospect of permanent arrest, and *per contra* the longer the intervals, the better is the prospect. A tendency for the periods between attacks to lengthen may be taken as a favorable indication. The young man whose case has been already cited is an example of this kind. Up to his twelfth year his asthma was frequent and severe, whereas since that time his paroxysms have, without any treatment, grown distinctly less frequent and less intense. His improvement is probably due to the fact that his asthma originated in early childhood.

The influence of a long duration of this disease has been spoken of in what was said above concerning age. As years go on, the sufferer from this distressing complaint may be said to establish an asthmatic habit or fixed impressionability of the nervous system and bronchi, which precludes complete recovery even though the cause be discovered and removed.

(*d*) *Structural changes* in the lungs and air-tubes. These have also been incidentally mentioned under age and hence it may only be emphasized here that in proportion as such alterations have taken place is the prospect of cure from asthma unlikely. Frequently recurring asthma at last leaves permanent congestion of the bronchi together with emphysema, so that the patient wheezes more or less all the time and but slight irritation is required to intensify his dyspnœa into a positive asthmatic seizure. Under such circumstances it is not reasonable to expect permanent arrest of his malady from any line of treatment.

(3) The influence of asthma upon the life prospects must be viewed from another standpoint than that of death during a paroxysm. The physician may be called upon to state what will be the effect of asthma on the duration of life. This must of course depend upon the frequency and severity of the attacks, upon the age at which the complaint develops and many other factors. It is a fact that asthmatics have reached a considerable age in spite of having suffered from their malady since early childhood. Salter mentions two patients of sixty-nine and seventy years respectively who had each been asthmatic for sixty-four years. Nevertheless, such instances are exceptional. The production of serious secondary effects in heart and lungs is apt to preclude the attainment of such advanced age. Consequently, in attempting to prognosticate the length of life one must take into consideration the likelihood of such changes being developed, and, *cæteris paribus*, the more intense, protracted and frequent the paroxysms, the more apt are secondary effects to be established early and to be pronounced.

Finally, when such changes have once been developed they render the tenure of life uncertain and will probably serve to shorten it materially. The asthma in such cases intensifies the emphysema and this increases the frequency of asthmatic attacks; thus a vicious circle is established which must necessarily abbreviate life. Moreover, when such a condition has been reached there is the added danger of death from intercurrent affections.

Treatment.—This must be considered under two heads: (1) the therapeutics of the paroxysm, (2) the management during the intervals with a view to preventing a recurrence or removing the bronchitis on which many cases of asthma seem to depend.

(1) **The Asthmatic Attack.**—Most sufferers from this complaint have some favorite asthma powder which long experience has proven to be of real service, and therefore when a physician is summoned it is because the old remedy has been worn out or because the unwonted duration and severity of the paroxysm have occasioned alarm. As a rule the precise nature of the malady is at once apparent; but, since it is quite possible for an attack of acute pulmonary œdema closely to simulate asthma, it is well for the practitioner to resort to some remedy which would be devoid of danger in either event.

Accordingly, the safest and at the same time the most efficacious agent is a hypodermic injection of morphine and atropine. The size of the dose and the wisdom of a repetition must be left to the judgment of the physician, but less than $\frac{1}{4}$ of morphine and $\frac{1}{100}$ of atropine would hardly be likely to afford relief. The addition of atropine is justified by the consideration that it not only augments the action of the morphine in a favorable manner, but that of itself it exerts a beneficial influence over the asthma. Such an injection usually relieves the dyspnœa sufficiently to permit of a few hours' sleep and this proves an inestimable boon.

A few whiffs of chloroform will, likewise, allay the attack in some cases, but the effect is apt to be transient. There is also the objection that this agent is not wholly devoid of danger. The fumes of amyl nitrite or a hypodermic of $\frac{1}{100}$ of nitroglycerin may prove efficient in some instances, especially if the pulse is small and tense in consequence of arterial as well as bronchial spasm. Nitrite compounds, however, are not invariably remedial and may occasion intense headache. Such was my experience with amyl nitrite in my first case of bronchial asthma. Not only was I chagrined over the failure of the remedy I had so confidently praised, but the patient had the additional discomfort of a splitting headache without any abatement of her asthma. I also learned that such a mishap is likely to shake the patient's confidence in a young physician quite disastrously.

Should the remedies above named not arrest or greatly mitigate the intensity of the paroxysm I would recommend a trial of pyridin or iodide of ethyl. A few drops of the latter may be poured upon a handkerchief and inhaled, but its effect is not lasting. Moreover, when it is employed too often or for too long a time, it produces the characteristic iodine eruption.

Pyridin, which is very volatile and possesses an odor very like that of sprouting potatoes, may likewise be inhaled from a handkerchief; or 20 to 30 drops may be poured into a saucer and allowed to disseminate their fumes through the atmosphere of a small closed apartment. The action of this agent in overcoming spasm is quite prompt, and under its influence the tense contracted pulse becomes soft and full *pari passu* with the lessening of bronchial spasm and cessation of dyspnœa.

In this connection it may be stated that observation of the results of medication in cases of asthma has led me to the conclusion that the characters of the pulse furnish a tolerably reliable guide as to the kind of medication likely to relieve in any given case. If the pulse is small and tense and not particularly frequent, it indicates a remedy that will overcome spasm, i. e., a vaso-dilator. If, on the contrary, the pulse is unduly frequent and compressible, although small, in consequence of cardiac exhaustion and dilatation, it indicates a heart stimulant and tonic. In such a case the right heart has become overpowered, pulmonary stasis has augmented the preëxisting hyperæmia of the bronchial mucosa and the sufferer is exhausted.

In this stage vaso-dilators are scarcely likely to be of much service; it is more rational to prescribe camphor, caffeine, digitalis, etc., while at the same time injecting a small dose of morphine, $\frac{1}{8}$ to $\frac{1}{4}$, to tide over the patient until the other remedies may have time to produce their effect.

Apropos of this subject it may be said that the relief derived by some asthmatics from whisky and tobacco is owing to their power of overcoming spasm of both bronchioles and arteries. Thus, I once knew an old asthmatic who always flew to his pipe and bottle of whisky as soon as his enemy attacked him. During his intervals of freedom he neither smoked nor drank, but when his asthma was upon him he endured an amount of whisky that would have intoxicated him at other times, and he could smoke the strongest kind of tobacco without becoming nauseated. The beneficial effect of tobacco smoke lies in the pyridin it contains.

The ability of an emetic, as ipecac and lobelia, to relieve an asthmatic attack or in some instances to prevent its occurrence, is owing to their vaso-dilator or antispasmodic action. To be efficient, however, they must be prescribed in doses that either will prove actively emetic or will keep the individual on the very verge of nausea. It is said by Hyde-Salter that 20 grains of powdered ipecac has been known to cut short a paroxysm of asthma in its incipiency.

Potassium iodide and belladonna have also been much used for the treatment of asthma and the latter in particular was highly vaunted by Trousseau. They have to be taken for a considerable time, however, and are not invariable in their beneficial effects. Accordingly, they seem to me less reliable than the agents previously mentioned.

There is a great number of asthma powders and most sufferers keep on hand some one which they have found effective. These all contain nitrate of potash and many of them stramonium. The following formula for such an asthma powder is taken from Fowler's work:

℞ Stramonii foliorum	ʒjv
Anisi fructus	ʒij
Potassii nitratis	ʒij
Tabaci foliorum	gr. v

M. Sig.: A teaspoonful of this powder may be placed in a saucer and ignited, and the fumes inhaled through a paper cone.

It is said that valuable information may be obtained by determining the presence or absence of dermographia. If the characteristic urticarial line be produced by firmly drawing the finger nail or the blunt end of a pencil across the skin, it indicates capillary paresis and points to hyperæmia of the bronchial mucosa. Under such circumstances speedy relief from the asthma is likely to follow if ⅛ of a grain of suprarenal extract is dissolved on the tongue every two or three hours. When, on the contrary, a white line remains on the skin, there is capillary spasm and vaso-dilators, as amyl nitrite or nitroglycerin, afford relief.

My experience has accorded with the truthfulness of the above statements. In one case in which dermographism was present the suprarenal extract brought cessation of the asthma after a time and also warded off other attacks. In other cases which did not show this abnormal irritability of the capillaries of the skin the remedy proved entirely useless.

Management During the Intervals.—This embraces the removal of the cause whenever possible, and the improvement of general health or of any complication as chronic bronchitis which may be aggravating the asthma. As regards the possible causes of the attack it is important to remember that brilliant cures have sometimes followed operative treatment of nasal defects. Consequently, if any condition exists which might initiate a seizure and, in particular, if a sensitive point be discovered the irritation of which occasions dyspnœa, it should receive appropriate treatment.

Digestive disorders or indiscretions should be corrected and in particular the asthmatic must be cautioned against eating too heartily in the evening. Constipation is to be corrected and menstrual disorders are to receive suitable therapeutic attention. If the individual is neurotic, as is not infrequently the case, every attempt must be made to improve the state of his nervous system.

In many instances there is more or less chronic bronchitis or emphysema which, if not actually responsible for the asthma, certainly contributes powerfully to its continuance. In all such instances the bronchial catarrh is to be combated along the lines laid down in the chapter dealing with that subject. In other cases it may be the heart, the blood-vessels or the kidneys, which must be treated in the hope of mitigating the attacks and lengthening the intervals between them.

Change of climate is in many cases the only means of freeing the asthmatic from his enemy. Some persons may find relief by removing only a short distance; but as a rule it is safer to advise residence in an entirely different climate. The mountainous regions of Colorado, New Mexico, Arizona etc. have brought permanent immunity to many an asthmatic and hence may be recommended except for such as have developed considerable emphysema of the lungs.

There is probably no complaint of the respiratory organs that will call for greater skill, ingenuity and patience on the part of the practitioner than bronchial asthma.

CHAPTER VIII

TRACHEAL AND BRONCHIAL STENOSIS

NARROWING of the air-tubes at some point in their course is a comparatively frequent occurrence, more frequent, in fact, than is clinically recognized. The explanation of such oversight lies in the fact that although tracheal or bronchial stenosis may, under certain circumstances, produce very characteristic symptoms, these are apt to be overshadowed by the manifestations of the primary affection.

Ætiology.—The causes are divisible into two great groups, (1) extrinsic and (2) intrinsic. In the former belong the cases in which the obstruction is owing to compression from without, while the second comprises cases which arise from disease of the tube or the introduction of a foreign body.

(1) **Extrinsic Causes.**—Compression of the trachea may result from (*a*) aneurysms, *e. g.*, of the transverse arch or innominate artery, (*b*) thyroid enlargement, (*c*) new growths, (*d*) mediastinal abscess.

The structure of the cartilaginous rings of the trachea, i. e., their failure to unite posteriorly at what is known as the membranous portion of the windpipe, permits this tube to be compressed with comparative ease by any tumor exerting lateral pressure. Hence it is that very considerable degrees of narrowing may be occasioned by aneurysms, hypertrophy of the thyroid, cancer, etc.

Aneurysm.—The relation of the windpipe to the transverse portion of the thoracic aorta is such that it can scarcely escape compression by aneurysm situated on this part of the arch. Indeed, a sac not larger than a walnut may be so located as to exert very grave pressure upon the trachea just above its bifurcation and yet declare its presence only by dyspnœa and hæmoptysis.

Thyroid Gland.—Enlargement of the thyroid gland is capable of producing very considerable degrees of narrowing. The accompanying figure (Fig. 10) shows what is styled a "saber sheath" trachea, occasioned by hypertrophy of the right lobe of the thyroid which had dipped down behind the clavicle and also pushed aside the innominate artery and its two subdivisions. The specimen was discovered by Dr. W. T. Eckley in the anatomical laboratory of the College of Physicians and Surgeons, Chicago, and, of course, was without previous history that could be obtained. When all three lobes of the thyroid are sufficiently enlarged they may grasp the trachea in front and at the sides and literally produce slow strangulation. Exceptionally a lobe or an accessory lobe may dip down toward the rear in such a manner as to compress the membranous portion of the windpipe and occasion very distressing dyspnœa.

The *new growths* which produce narrowing of the trachea are usually malignant and have developed in the mediastinum or the œsophagus. The intimate association of the gullet with the noncartilaginous portion of the trachea enables a comparatively small tumor in the wall of the former seriously to compromise the caliber of the latter.

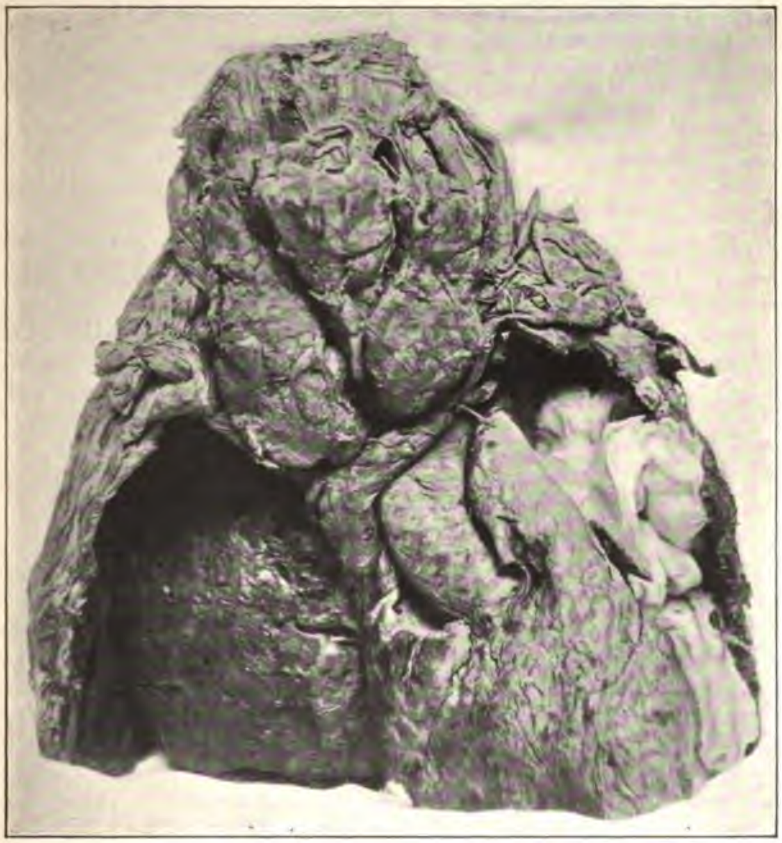


FIG. 10.—"Saber sheath" compression of the trachea by thyroid tumor.

Mediastinal Abscess.—Compression of the trachea by abscess within the mediastinum is rare, yet cases have been recorded (Schnitzler). West cites such a case in a lad of nine years who was under treatment for caries of the dorsal vertebræ. Dyspnœa was a very distressing feature which developed some time after he had been put to bed on his back. At the autopsy it was found that an extensive collection of pus, which evidently owed its origin to the spinal caries and occupied the mediastinum from the level of the ninth to the second dorsal vertebra, had so pressed upon the trachea as almost to obliterate its lumen just above the bifurcation.

THE EXTRINSIC CAUSES OF BRONCHIAL STENOSIS include those that have been mentioned with reference to the windpipe and a number of other conditions which for anatomical reasons cannot affect the trachea.

(*a*) *Lymph Glands.*—Among the commonest causes of bronchial compression are enlarged peribronchial lymph glands. These are grouped about the roots of the lungs in such a manner that when enlarged from tuberculous disease, (by far the most frequent cause of their increased size,) they can scarcely fail to produce more or less pressure upon the bronchus. West expresses the original view that peribronchial nodes may undergo transitory swelling in the course of bronchitis and other inflammatory complaints of the lungs, and hence may occasion sufficient constriction of a main bronchus to give rise to temporary dyspnœa with stridor. The opinion is ingenious but scarcely capable of demonstration.

(*b*) *Aneurysm* of the aortic arch is a very frequent cause of bronchial stenosis. A sac springing from the ascending arch may, when it develops backward, seriously obstruct the right bronchus, whereas the left bronchus is likely to be stenosed by aneurysm of the transverse or descending portion of the arch. Exceptional cases have been reported however of compression of the left tube by a sac arising from the ascending portion. A large aneurysm involving the entire arch may exert pressure on both the trachea and its two divisions.

(*c*) *Malignant tumors*, whether of the mediastinum, lung or œsophagus, not infrequently occasion bronchial stenosis. Hoffmann states, however, with regard to œsophageal cancer that it is more apt to occasion perforation than simple compression of a bronchus.

(*d*) *Mediastinal abscess*, whether it be from acute mediastinitis or the result of caries of a bone, as vertebra or clavicle, may also produce bronchial narrowing and instances have been recorded.

instances of bronchial stenosis. In one the left main tube was occluded by what the doctor took to be a "submucous fibroma" while in the other case the left main bronchus was obstructed by a simple cyst.

Primary carcinoma was the cause of stenosis of the trachea and main bronchi in a case observed by Langhans. The new growth had originated in the submucous glands. This case is especially interesting since malignant tumors of the bronchi are generally secondary.

(*c*) *Syphilis.*—This is usually in the form of ulceration that has led to loss of the cartilage at a given point and subsequent contraction. It is stated, however, (Hoffmann) that syphilitic peribronchitis may also occasion narrowing of a bronchial tube. Syphilitic ulcer is a quite common cause of tracheal stenosis of which a considerable number of extreme examples have been reported. It goes without saying that stenosis of the air-tubes from this disease occurs only in the late stage.

(*d*) *Ulceration of other nature* from tuberculosis or trauma, is also said to be capable of narrowing the lumen of a bronchus, but, as Hoffmann pertinently remarks, reliable observations on this point have not been recorded.

(*e*) *Perichondritis* may lead to partial occlusion of a bronchus by the formation of abscess or by necrosis of cartilage and the subsequent contraction of that portion of the bronchial wall that has suffered from loss of tissue.

(*f*) *Simple inflammatory swelling*, which, according to older authors, is capable of producing bronchial stenosis by extension of inflammation from the trachea, can hardly lead to such a degree of stenosis of the main tubes as to be manifested by clinical symptoms. It is, however, as stated by Ziegler, a common cause of narrowing and even occlusion of bronchioles in the course of acute and chronic bronchitis and it may be of more frequent occurrence than is recognized, for tubes of the third and fourth dimension to be thus stenosed. The clinical effects are, however, so merged in those of the bronchitis as to form but a part of them and belong essentially to that disease.

Under the term "saber sheath" trachea, Simmonds has described a form of tracheal stenosis sometimes occurring in men past fifty years of age. The cartilaginous rings become ossified and flattened laterally so that the tube is narrowed in its transverse diameter. The cause appears to be the ossification of the rings and must be explained by the tendency of cartilage in general to undergo such change with the development of senility. An example of bronchial stenosis from a similar ossification of the cartilages has been described by Gerhardt under the term Bronchiostenosis Ecchondrotica (Hoffmann). The patient, a male, had suffered from attacks of bronchitis and at the necropsy the rings of the left main bronchus and of numerous of its subdivisions were discovered thickened and on their inner aspect covered by a thick layer of bony substance. In places, moreover, exostoses, having a branched and wartlike arrangement, still further encroached upon the lumen of the tubes.

Morbid Anatomy.—The *direct* effect of compression of an air-tube from without is an inflammatory swelling of the mucous membrane opposite the point of pressure. In time still graver nutritional disturbance results in ulceration, which in turn may be succeeded by perforation. This is especially apt to be the case when the compression is due to a tumor or aneurysm.

The *indirect* or remote effects are seen in changes that take place in the lung. Peripheral to the stenosis there is a tendency to atelectasis more or less pronounced and extensive, according to the degree and seat of narrowing. There is, furthermore, as was pointed out in the chapter on bronchiectasis, a tendency to bronchial dilatation and its consequences. Infection of the area supplied by the constricted tube may lead to broncho-pneumonia. If some one of the smaller tubes is narrowed the portion of the pulmonary parenchyma which lies internal to the stenosis is said invariably to become emphysematous (Hoffmann) in consequence of paralysis of the bronchial muscles and the accumulation of secretions.

The opposite lung undergoes compensatory emphysema the degree of which is determined by the seat and degree of the stenosis. Furthermore, the embarrassment of circulation within the atelectatic lung may, when the stenosis abruptly occurs, be so great as to throw more strain on the vessels of the sound parts than they can well bear, and thus lead to serious even dangerous pulmonary œdema. Finally, the sudden stenosis occasioned by a foreign body may lead to pneumonia and abscess or gangrene as will be mentioned in the succeeding chapter.

Symptoms.—These depend upon the seat and degree of the stenosis and the rapidity with which this is developed. Narrowing of the trachea or of the two main branches occasions serious interference with respiration which may, under some circumstances, amount to actual strangulation. The *dyspnœa is inspiratory* and compels the patient to bring all his auxiliary muscles into play. He sits with head bowed somewhat forward while the scaleni and sterno-mastoids stand prominently forth with each labored attempt to force air past the point of narrowing. The face is anxious, the nostrils alternately dilate and contract, the epigastrium and soft parts of the chest and neck retract with each inspiratory effort. There is stridor, and the voice is feeble and bleating. Examination of the chest discloses evidence of the obstruction to ingress of air in the feebleness of the breath-sounds, along with persistence of pulmonary resonance.

If the bronchus of one side only is affected dyspnœa is less marked since the opposite lung is usually sufficient for the needs of the system so long as the patient is at rest. As soon, however, as he makes exertion, the respiration at once becomes embarrassed. If the stenosis is abruptly induced the sudden diminution of respiratory capacity occasions more decided shortness of breath than when the slow development of stenosis affords the system time to accommodate itself to the altered condition.

Dyspnœa is the chief symptom, however, in all cases in which a tube of considerable size is constricted. It may in some instances assume characters very similar to those of bronchial asthma.

Stridor is generally present and may be accompanied by a palpable vibration over the seat of the stridor. In tracheal stenosis these phenomena are perceived over the lower portion of the tube behind the upper part of the *manubrium sterni.* In stenosis of a main bronchus, stridor and possible thrill are apparent at the root of the lung, commonly in one or the other interscapular regions and very likely also at either side of the sternum at about the level of the second intercostal space.

Pain is not a symptom unless the compression be due to aneurysm or malignant tumor, and even then is apt to be a dull painful feeling rather than acute. *Cough* and *expectoration* are present only when bronchitis is associated or some of the pathological results supervene which usually occasion such symptoms.

The disease in question may pursue a slow or a rapid course according to the nature of the primary affection and the severity of the local and consecutive changes. In aortic aneurysm and in new growths the course of the primary affection determines the duration of the case. In stenosis of a bronchus from pressure by an enlarged peribronchial gland the symptoms may be slight and the course exceedingly protracted.

Physical Signs.—Whether or not stenosis of the air-tubes gives rise to objective symptoms depends upon its cause as well as upon its seat.

In cases of aortic aneurysm or of mediastinal or intrathoracic tumors the phenomena pertaining to the primary disorder are apt to overshadow and obscure those belonging to bronchial obstruction when this exists. For example, aneurysm of the transverse and descending portions of the arch may compress the left main bronchus, but is also likely to interfere by pressure with the function of the left recurrent laryngeal nerve. When this nerve is irritated and not paralyzed attacks of almost suffocative dyspnœa take place in consequence of spasm of the adductors of the vocal cords. There may be diminished respiratory capacity and physical signs due to compression of the bronchus, but these symptoms sink into insignificance beside the suffocative attacks indicative of pressure on the nerve.

When, on the contrary, the stenosis is occasioned by some condition, e. g., enlarged lymphatic glands or syphilitic cicatrices, which first declares itself by symptoms of tracheal or bronchial obstruction, objective signs are likely to be well marked.

Inspection at once detects signs of dyspnœa which, because of recession of the supraclavicular regions, the intercostal spaces and the epigastrium, is seen to be inspiratory. It is thus plainly apparent that the difficulty is in getting air into and not out of the lungs. The sufferer assumes a more or less upright posture and inclines rather than extends his neck as when the obstruction is laryngeal. If the stenosis involves the trachea just above its bifurcation or both primary subdivisions as may be the case in sarcoma of the anterior mediastinum, of which I have observed an example, both lungs are affected and the entire chest displays signs of inspiratory obstruction. When
bronchus is involved, then the signs of deficient lung-expansion are
to the corresponding half of the thorax. In such an event the
side is likely to display exaggerated respiratory movements.

tion is not apt to furnish information that cannot be obtained by
ans of investigation. It may, however, enable one by perceiving
on of maximum stridulous vibration to locate the seat of the

Mensuration is of service in cases of unilateral stenosis in establishing the fact that the affected half of the thorax measures less than the other and thus corroborates the information derived by inspection. It is not always easy by the eye alone to determine positively that one side is of smaller size.

Percussion.—Pulmonary resonance is more or less impaired below the seat of stenosis. If the trachea is involved, both lungs are deficient in resonance, whereas, if one main bronchus is obstructed the percussion over that lung is defective as compared with its fellow. Pronounced dullness is not likely to exist unless some of the secondary changes are present, i. e., collapse, broncho-pneumonia, etc. If bronchiectasis exists the percussion-findings are those described in that chapter.

Auscultation.—The most marked feature is stridor which may be so loud and vibrant as to obscure all other sounds. It is usually found to have its maximum intensity in the upper part of the chest over the bifurcation, or in the back on one side close to the root of the lung. Should the stenosis not be so extreme as to occasion stridulous respiration, the breath-sounds will be found diminished in the parts below the seat of constriction.

Diagnosis.—Under some circumstances this may be extremely easy, while in other cases it cannot be more than inferential. The combination of inspiratory dyspnœa and stridor, with diminished but not abolished resonance, can scarcely leave room for doubt. In obscure cases attention must be paid to history and symptoms of respiratory embarrassment, and careful search must be made for signs of tumor, aneurysm, previous syphilis and enlarged lymph nodes in the so-called bronchial gland area, i. e., in the interscapular regions at the level of the fourth dorsal spine (Fig. 11). In the event of failure to discover distinctive physical signs tracheoscopy and bronchoscopy should be resorted to, in the hope of detecting a syphilitic cicatrix or visible narrowing of the trachea above its bifurcation or of one of its main branches.

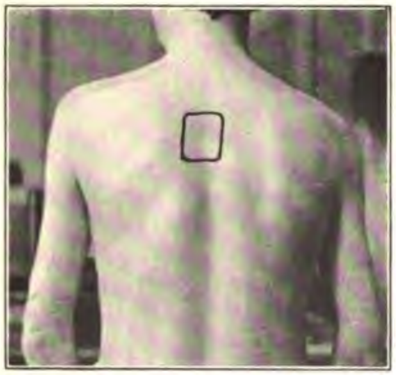


FIG. 11.—Diagram showing location of "bronchial gland area."

Furthermore, diagnosis to be complete should include not only the determination of the existence of stenosis, but also its seat and the nature of its cause. Should bronchoscopy fail to locate the obstruction, then minute comparison should be made of the two halves of the thorax with a view to detecting inequality, no matter how slight, in their respiratory play and the relative intensity of their breath-sounds. The cause may be ascertained by the history, e. g., of syphilis or possibly of the inhalation of a foreign body, and by the discovery of general symptoms pointing to aneurysm or malignant disease.

Differential Diagnosis.—(1) Laryngeal Stenosis.—In this affection the inspiratory dyspnœa is accompanied by extension of the neck and the up-and-down movements of the larynx. Laryngoscopy is likely to disclose the seat of constriction. (2) Bronchial Asthma.—(*a*) Dyspnœa is chiefly expiratory. (*b*) The chest is expanded. (*c*) The percussion-note is hyperresonant. (*d*) Excursion movements of the diaphragm are abolished and the lung-borders are fixed. (*e*) The chest is filled with sibilant râles and there is no inspiratory stridor. (*f*) There is history of previous attacks often extending through a period of years.

Prognosis.—For the most part this is unfavorable. Excepting in cases of stenosis due to a foreign body or to a syphilitic ulcer there is no likelihood of relief by treatment. Cases originating in aneurysm or malignant neoplasms are certain to progress and ultimately prove fatal, either through strangulation, destructive changes in the lung, or the effect of the primary disease upon the general health. In these cases perforation of the compressed bronchus is a possibility which should be borne in mind, since it may be attended by profuse, even fatal, hæmoptysis or other distressing symptoms. On the other hand, some cases of tracheal and bronchial stenosis may persist for years, depending on their cause, and the patients may die from intercurrent disease.

Treatment.—In the majority of instances this is limited to the alleviation of suffering by morphine, heroin, etc. If the stenosis is due to syphilis, mercury and iodides are indicated, but can scarcely be expected to influence old cicatrices. When the trachea is the seat of stenosis, or, indeed, when this is situated in one of the main bronchi, attempts may be made to dilate the stricture by means of rubber sounds carefully introduced through the larynx. Von Schroetter is said to have resorted to this method with gratifying results in cases of syphilitic obstruction. In resorting to the sound, however, one must be very sure that the stenosis is not the result of aneurysm, since perforation of the sac might be produced.

CHAPTER IX

FOREIGN BODIES IN THE BRONCHI

THE frequency with which foreign bodies gain access to the air-tubes is such as to make the subject by no means unimportant. The mode of occurrence is through inhalation and the substances thus inhaled are of the most varied sort. Strictly considered this subject should include the aspiration of fluids or particles of food in consequence of laryngeal paralysis or diseases of the nervous system and the escape of pus, secretions etc., during operations on the throat or anæsthesia; but as such mishaps occasion so-called aspiration pneumonia, these occurrences will be dealt with in another place.

Almost every sort of foreign body small enough to pass through the chink of the glottis has been known to gain access to the larynx, trachea or bronchial tubes. The list includes beads, buttons, pins, needles, nails, coins, a small bolt, beans, grains of corn, seeds, heads of grain, grasses, pieces of straw or bone, a cork, fish bones, a fragment of a clam shell, the dart of a blowgun and even a toy whistle. As might be expected from the inveterate habit of children of putting all sorts of articles in the mouth, this accident occurs most often though by no means exclusively among children.

The mishap has been known to take place during sleep, but it usually occurs while the child is at play when, in consequence of a laugh, a sudden start or a cough, he makes a quick vigorous inspiration which draws the object from the mouth through the open glottis into the parts below.

Morbid Anatomy.—The pathological results vary much as determined by (1) the part of the respiratory tract in which the offending body lodges, and (2) by the size and nature of the inhaled object together with the character of the germs introduced. If the foreign body happens to rest in the larynx or trachea or in its passage produces irritation, it may in fortunate cases occasion an almost instantaneous reflex cough by which it is at once expelled. Unfortunately however such is not always nor indeed often the case.

The object passes further downward and enters one of the bronchi. In the majority of instances it passes into the right main bronchus. The reason for this is variously given by writers. All agree in stating that it is due to the greater size of the right as compared with the left for one reason (Fig. 12), but in addition some (Lenhartz, e. g.) say this course is determined by the fact that the right tube descends more vertically (*steil abwärts*) than does the left.

English authors, on the contrary, following the statements of Stokes and

Gray, (e. g., West and W. Fox) assert that the right bronchus is given off at a more nearly right angle and that a body passing down the trachea is deflected into the right main bronchus because the sæptum deviates slightly from the median line toward the left. This discrepancy of statements led me to inquire concerning the real facts of W. T. Eckley, Professor of Anatomy at the Chicago College of Physicians and Surgeons (Medical Department of the Illinois State University). Dr. Eckley stated that he had made sixty-five dissections for the special purpose of determining the comparative size and relations of the two main divisions of the trachea, and accordingly he was kind enough to send a statement which may be summarized as follows:—

It must be conceded that the caliber of the bronchi is larger on the cadaver than in life, and his sixty-five cases show the size of the right as compared with that of the left main bronchus to be as 1 to .75. In seventeen cases examined, in which one lung had been practically rendered useless through disease, the bronchus of the competent lung was equal in size to that of the trachea. The bronchus of the diseased lung was much reduced in size, being practically but a vestigial conduit. In practically all of his sixty-five cases the declivity of the right bronchus was greater than that of the left the difference being due to the position and attachment to the trachea of the œsophagus which as shown by the accompanying diagram lies to the left of the median line and behind the left bronchus (Fig. 12).

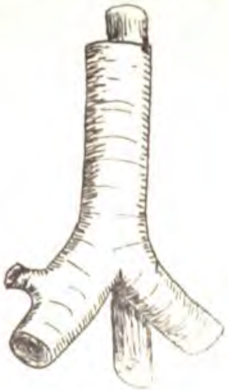


FIG. 12.—Diagram showing relations of trachea, bronchi, and œsophagus.

In consequence of this attachment the trachea and left bronchus are elevated by the peristaltic movements of the gullet excited by the presence of a foreign body in the oropharynx. If on the cadaver the œsophagus is distended by paraffin the elevation of the left and the declivity of the right bronchus are increased. If now a bullet is dropped into the trachea, as he had done in 200 experiments, it finds its way into the right bronchus more often than into the left, regardless of the condition of the lungs and their effect on the bronchi—108 times into the right and 92 times into the left.

The position of the sæptum can exert no influence in determining the direction of the foreign body, since upon deep inspiration the sæptum becomes flattened to the extent of becoming practically planiform. Consequently the factors that determine the direction which a foreign body will take must ater caliber and the declivity of the right bronchus, in consequence there is a more forcible current of air entering this tube.

ckley conceives that if the presence of the foreign body should, rs the trachea, excite peristalsis in the œsophagus, this peristaltic ld, by raising the left bronchus, render the body more likely to enter ite branch of the windpipe. Such an explanation seems scarcely in my opinion, since the foreign body enters the larynx during inspiration when, as is said, this structure is "off its guard," and hence a reflex attempt at deglutition should not be excited. It seems far more likely that the influence which the condition of the lung exerts on either bronchus, as regards its size and direction, is a determining factor of greater importance than has been recognized.

If the foreign body is of the size and shape to occlude a main bronchus it at once occasions symptoms of asphyxia which may even prove fatal. If its shape is such as not to plug the tube completely, or if, by reason of its smaller size, it passes into a bronchus of the third or fourth dimension, it may lead to changes in the tube or surrounding structures. These changes depend upon the composition of the object, upon the injury directly sustained by the mucous membrane on which it rests, and upon the nature of the accompanying germs. If a smooth, round body, as a marble, is not driven tightly into the bronchus it may become dislodged by the act of coughing and be tossed upward into the windpipe whence it again rolls into the same or the opposite air-tube. More often the foreign body becomes impacted and gives rise to local irritation or inflammation, that is, to bronchitis and bronchopneumonia. If it possess sharp edges or corners, these lacerate the delicate mucosa and thus prepare it for the implantation and growth of bacteria which accompany the invading body. The infection thus induced may lead to ulceration of the bronchus with or without pulmonary abscess or gangrene, a by no means rare result.

In other cases the resulting changes are of a chronic nature. Instead of an actively destructive process there is a slow inflammation which leads to chronic bronchitis and interstitial pneumonia with the subsequent development of bronchiëctases. Godlee states that bronchial dilatation is the most frequent result. In this manner cavities are formed which give rise to symptoms of chronic phthisis.

Aside from the local injury resulting directly or remotely there are often serious effects exerted on parts of the lung beyond the seat of the foreign body. If this but partially occludes the bronchus, air may enter the bronchial tract beyond, during the active force of inspiration, but be unable to escape in like measure during the less powerful expiratory act, and thus emphysema may be induced. If on the contrary the plugging be such as wholly to prevent the entrance of air, atelectasis results in that portion of the lung supplied by the occluded bronchus. In time, if the plug be not removed, this collapsed area undergoes fibrosis and bronchiectasis.

The development of fibrosis is in accordance with the physiological law that if parenchymatous tissue remains sufficiently long unused, it undergoes transformation into fibrous tissue. As the tendency of such newly formed tissue is to contraction, the walls of the bronchi are drawn away from each other and dilatations result. There is also the influence pointed out by Hoffmann of bronchial stenosis in the production of bronchiectasis, viz., inflammatory change in the bronchial wall and the distending force of air pressure.

The collapsed area may become infected and undergo acute pneumonia with abscess or gangrene. An abscess may rupture into the pleural cavity and occasion fatal empyema (Fox), or the empyema may result from the foreign body's eventually making its way into the pleural cavity. West cites the remarkable case recorded by Carpenter in which a set of false teeth which had been swallowed thirteen years previously was found in the pleural cavity where it had caused empyema. Fox states that foreign bodies have been known to travel from the bronchi through the lung and to appear beneath the integument, having even penetrated the chest-wall.

An abscess of the liver as well as of the contiguous portion of the lung has been known to result. Pericarditis and mediastinal abscess have been reported, and the penetration of the body into the mediastinum has caused cutaneous emphysema of the neck and trunk. In one case a foreign body made its way into the colon but was subsequently recovered through the mouth.

It is not necessary for pulmonary abscess to be confined to the lung originally penetrated by the foreign body, for through aspiration of sæptic secretions a suppurative inflammation of the opposite lung may occur. Tuberculosis may apparently as a direct result, follow the inhalation of a foreign body around the part where it lodges or tuberculization of both lungs may take place after the lapse of years.

Hæmorrhage is a very possible occurrence when the inhaled object is a sharp pointed one or has rough edges capable of lacerating the bronchial wall. Rokitansky observed a memorable instance of this kind. A dart of a blow-gun entered the left main bronchus and lay in such a position that during a coughing fit its point pierced the opposite wall of the trachea and innominate artery with, of course, fatal hæmorrhage on the twelfth day after the original mishap.

Symptoms.—These vary greatly in different cases and may be considered under two heads, (1) those that attend or immediately follow the accident and (2) such as are attributable to the more remote pathological consequences.

(1) The symptoms immediately following the entrance of the foreign body are determined (*a*) by the size and other characters of the body itself, (*b*) by the site of its lodgment, and (*c*) by its firm impaction or its mobility in the bronchus which it enters. More or less *cough* is excited by the passage of the body through the glottis and into the bronchus. If the body is small and enters a tube of the third or fourth order whose sensibility is slight, but trifling reflex irritation may be occasioned and cough may not be marked. Indeed, cases are on record in which so little disturbance resulted that it was not quite certain even in the patient's own mind whether he had or had not inhaled the article which was in his mouth but a moment before. Generally, however, there is violent and spasmodic cough which may even persist for days or weeks and prove most distressing.

nœa is a symptom of more or less prominence in most cases, and when
bronchus is occluded may assume truly alarming intensity. The pa-
t only appears, but may actually be, in peril of immediate suffocation.
ggles for air, his face becomes livid, his eyeballs start from their sock-
uxiliary muscles of respiration tug violently, he clutches his throat
ws himself about, crying out that he is going to strangle, or he writhes
less agony. Bloody froth may issue from the mouth or he may speed-

ily lose consciousness. If at this time the chest is examined it is found that over one lung the breath-sounds are absent while over the other they may be exaggerated. The affected half of the thorax may display impaired motion or may even appear retracted. The heart throbs violently and the soft parts sink with inspiration.

If one of the smaller bronchi is the seat of the foreign body, there may be evidence of abolished function of a circumscribed area with rapidly developing loss of resonance or, on the contrary, hyperresonance according to the completeness with which the tube is occluded. Fortunately for the patient, the lodgment of the body in bronchi of the third or fourth order is not attended with such distressing dyspnœa, although the patient is very likely to experience some embarrassment of breathing or to be conscious of a sense of distress or fullness in that part of his lung.

Pain is not usually complained of and yet if the foreign body is of such a nature as to pierce or otherwise wound the bronchial mucosa it may occasion pain. As stated above the sensation is more apt to be one of weight or oppression that is akin to pain rather than actual suffering of this nature.

A small non-irritating substance may penetrate deeply and, aside from producing more or less cough and a momentary sense of some foreign object having passed into the lung, may not cause the patient especial discomfort. In such an event the symptoms are likely to appear with the subsequent development of broncho-pneumonia and then form a part of that affection. Such a local pneumonia may, according to Hoffmann, make its appearance and be detected by physical signs of dullness and abolished respiratory sounds within twenty-four hours from the occurrence of the accident.

Very rarely the foreign body may not become impacted within the bronchus but may be dislodged by the cough and, being cast upward, may find its way into a neighboring tube of the same lung or into the bronchi of the opposite lung. The patient may then be conscious of the migration of the object, and the physical signs of its presence may shift about in such a manner as to betoken the mobility of the substance.

Irritation of the bronchial mucosa is likely to be shown by more or less expectoration attending the cough. Andrew is said to have observed the case of a child who inhaled a fragment of a nutshell into the right bronchus and, after having had a thin muco-purulent sputum for a few days, finally expectorated the offending material.

(2) **The secondary symptoms,** as they may be termed, or those that result from the pathological consequences, necessarily depend upon the nature of these consequences. If broncho-pneumonia ensue, there are the high and often irregular fever, rapid pulse, accelerated breathing, cough, expectoration and signs of local induration. If abscess or gangrene develop the sputa assume a purulent or fœtid character, there is intermittent or sæptic pyrexia with sweatings and prostration. In short the clinical picture is that of septicæmia and, unless operative measures are resorted to with successful removal of the offending body, or unless this is expectorated along with the sputa, death is the probable result.

In other less severe and dangerous cases the symptoms become those of a chronic phthisis or of bronchiectasis, i. e., cough with purulent expectoration, perhaps containing shreds of pulmonary tissue, growing emaciation, loss of strength and fever which, in its character and intensity, depends upon the rapidity and extent of the lung destruction. In such cases the symptoms are so like those of chronic pulmonary tuberculosis, and the history of the inhalation of a foreign body may be so remote that the cases may be mistaken for a primary instead of a secondary tuberculosis which they in reality are.

In rarer cases the clinical picture may be that of empyema or mediastinal abscess, with or without subcutaneous emphysema, or, in protracted cases with unreliable history, the symptoms may indicate a deeply seated and so obscure a pulmonary mischief that the physician may not be able to locate the exact seat or nature of the disease.

The effects of the lodgment and retention of a foreign body in the bronchus are well shown by the following case taken from the records of Cook County Hospital, Chicago: E. B., a Belgian cook, aged fifty-eight, was admitted to the hospital September sixth, 1903, having been ill six weeks and complaining of inability to swallow anything but liquid food. Solids seemed to lodge about the middle of the sternum and cause vomiting. He had headache, pain in the region of the liver, nocturnal chills and perspirations. He was short of breath the last three weeks before entering the hospital. Three years before, patient was in the hospital with severe cough and since that time has coughed and expectorated purulent material.

The physical examination showed a muscular, well-developed man; pulse weak and irregular; slight icterus of the sclera; breath foul; chest normal in contour and of moderate expansion; lungs resonant throughout but breath-sounds harsh and wheezy; numerous moist and whistling râles over the whole of both lungs, most marked on expiration which was somewhat prolonged; vocal fremitus not increased; heart slightly enlarged to left and the sounds very weak, an uncertain aortic systolic murmur; liver palpable two inches below arch of ribs and border firm and smooth.

The temperature ranged from normal to 100.5° F. respirations 24 to 30 for three or four days but then fell to normal and so remained until his discharge. The urine was that of a mild interstitial nephritis; sputum contained no tubercle bacilli. An exploration of the œsophagus with a sound showed an obstruction at the level of the bifurcation of the trachea and another at the cardiac orifice of the stomach. Patient was discharged improved September 28th, 1903.

The 28th of following December he returned and remained until May 9th,
1904; complained of the same trouble, pain in the region of the liver, in the
shoulder and beneath the sternum; loss of flesh and increasing weakness
g his stay; occasional periods of slight fever; cough constant with puru-
xpectoration; urine findings as before. During this stay in the hospital
rostomy was performed and thereafter the patient was fed through a

y 9th he was again discharged only to return for a third time on June
04 with slight fever, rapid respirations, lungs full of larger and smaller

moist râles, expiration prolonged; died June 11th, 1904. Autopsy by Dr. W. A. Evans June 15th.

For the sake of brevity, only the findings in the lungs and œsophagus are here given. There was a stricture of the œsophagus 1 inch in length and the diameter of the gullet at this point was reduced to $\frac{1}{8}$ inch; enlarged mediastinal lymph glands, not metastatic, laryngitis, tracheitis and bronchitis; in the right main bronchus a ten-cent piece lying in a bronchiectatic cavity 1.5 cm. in diameter, which had fibrous walls and infringed on the œsophagus at the point of constriction; chronic interstitial pneumonitis and bronchitis of the right lower, of the middle and of the lower part of the upper lobe; emphysema of the upper lobe and pneumonitis of the apex; (left lung) emphysema, œdema, bronchitis and large lymph glands at the base but no metastases therein; right pleural adhesions and few left pleural adhesions with some fluid. Both the aorta and pulmonary artery were dilated.

The foregoing case is interesting on several accounts. In the first place there was absolutely no history of the aspiration of a foreign body into the lung, not even at the time of his first admission to the hospital when his cough was so severe. In the second place the coin had lodged in the right main bronchus and set up dilatation, precisely the condition said to result most often. In the third place, and this is the most interesting, at the point where the dilated and fibrous bronchial wall impinged upon the œsophagus a malignant growth had developed which on microscopic examination proved to be a carcinoma. It was this cancer which so greatly reduced the man in strength and yet he died probably of the acute pneumonia of the right lung together with the widespread bronchitis. It is likely that all the changes in the lower right and middle lobes (chronic interstitial pneumonia as well as bronchitis) were indirectly traceable to the foreign body.

Physical Signs.—**Inspection.**—The objective phenomena depend upon (1) the size and location of the foreign body and (2) the resulting pathological changes. If, owing to the smallness and deep penetration of the body, but a limited area of the lung has its function abolished, the patient may display no visible sign of dyspnœa, but if, as is more often the case, the offending material plugs a main bronchus or one of the second or third order, there is likely to be ocular evidence of distress. The interference with the free ingress of air is shown by the patient's struggle for breath and, upon inspection of the chest, it may be observed that the play of the two halves is unequal. The expansion of the occluded lung being deficient or wholly wanting, the corresponding side may show more or less retraction with each inspiratory effort.

In most cases inspection of the chest furnishes no reliable information as to the seat of the foreign body and an attempt must be made to discover its location by bronchoscopy after the method of Killian. Great dexterity is required, but the physician just named claims by means of long wide tubes properly introduced and illuminated to have seen as far down as into the bronchi of the second and third order and to have observed the branching of the main bronchus in the upper lobe. For details concerning bronchoscopy the reader is referred to works on Laryngology.

Palpation is of no special aid in determining the fact that a foreign body has actually been inhaled. The abolition of tactile fremitus over a circumscribed region of one half of the thorax while the parts surrounding retain such vibration might enable one to detect the seat of the foreign body when, through the history, it was ascertained that such a mishap had taken place.

Percussion may or may not be of aid in determining the fact of the aspiration of a foreign body and in locating its place of lodgment, according to the immediate effect produced. If partial occlusion of a bronchus leads to localized or unilateral emphysema, there will be hyperresonance over a limited region or throughout the entire lung. On the other hand, should atelectasis occur, this fact may be shown by dullness. These changes in the percussion-note, when associated with the changes to be described under auscultation, are of very great significance.

Auscultation.—If a main bronchus is the seat of the foreign body and the entrance of air is prevented, this fact is shown by absence of breath-sounds over the corresponding lung. If a small tube is occluded there may be absence of respiratory murmur over a circumscribed area, but if the affected region is deeply situated as well as of limited extent, auscultation may be wholly negative.

In some cases inspiratory stridor may be heard over the upper part of the chest or râles may develop of a rhonchal or bubbling character due to collection of secretion of the tube beyond its point of partial occlusion. In a unique case recorded by Angelis and cited by Lenhartz and Hoffmann a toy whistle which had lodged in the right bronchus emitted with each respiratory act a whistling sound that could be heard at a distance of 15 meters from the patient's chest.

For the physical signs belonging to the various pulmonary affections secondary to the lodgment of a foreign body in the bronchi, the reader is referred to the appropriate chapters.

Diagnosis.—Two facts must be determined if possible before the diagnosis of a foreign body in the lungs can be considered complete: (1) The fact that a foreign body has entered the bronchi, and (2) the place where it has lodged, with a view to its removal by surgical means if necessary.

The fact of the accident can generally be determined by the anamnesis and the symptoms of cough and dyspnœa which immediately followed the disappearance of some article that was in the mouth. The conjunction of history and symptoms usually makes it safe to rely upon the statements of the patient or of his friends, since, as pointed out by Fowler, the mishap is likely to make so profound an impression that he is not liable to be mistaken or to forget it. Should the history be indefinite or unreliable then diagnosis must be based on
inical findings that have been described.

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tory expansion of one half of the thorax with enfeebled or abolished
ounds over the corresponding lung and either hyperresonance or dull-
pending upon the degree of occlusion of the tube, form a combination
e of the lodgment of the body within a main bronchus. The penetra-
the inhaled substance to a deeper part, as into tubes of the third or

fourth order, is attended with less urgent dyspnœa or even none at all and occasions modification of the physical signs over a circumscribed portion of one lung, more often the right. Consequently, painstaking search should be made for the discovery of an area in which, with or without alteration of the percussion-note, the respiratory murmur is plainly diminished or entirely absent.

Should conclusive physical signs be wanting immediately after the accident the site of the foreign body may be revealed some hours later (twenty-four hours according to Hoffmann) by the development of dullness, absence of breath-sounds and other evidences of secondary pneumonia. The detection of such findings, therefore, over a limited area, corroborates the history and indicates the location of the foreign body.

Should the character of the symptoms and examination of the chest not enable one to ascertain the situation of the body, resort should be had to bronchoscopy after the method of Killian of Freiburg. Indeed such inspection is advisable in every case for corroboration of the results of other means of examination and with special view to removal of the foreign body through the larynx. Killian employs tubes of unusual length and diameter which are introduced as far down as possible and are brilliantly illuminated.

The X-ray may be employed for the location of the foreign body and, when the composition of the latter is suitable, should yield satisfactory results.

Differential diagnosis must, in the absence of reliable anamnesis, be made between the accident we have been considering and the following affections: (1) Laryngeal obstruction, (2) Tracheal or bronchial stenosis from external pressure or cicatricial narrowing, (3) Bronchial asthma.

In suddenly induced laryngeal obstruction there is (*a*) no history of the accidental inhalation of some article previously in the mouth, while there is quite likely to be a history of antecedent disease, e. g., diphtheria, that may have led to the formation of a false membrane in the larynx. (*b*) The inspiratory dyspnœa is attended with stridor and extension of the neck in the attempt to get air. (*c*) There may be fever or other symptoms of a primary process that has led to the obstruction. (*d*) Finally, laryngoscopy should clear up the diagnosis.

For the diagnosis of the other affections the reader is referred to the respective chapters.

Prognosis.—This must depend not alone upon the size and seat of the foreign body, but even more upon its condition as regards sepsis or asepsis. The arrest of a large body at the bifurcation or in the beginning of a main bronchus may, as already stated, cause perilous dyspnœa or even fatal asphyxia if not speedily removed. Whereas the penetration of the substance into deeper parts may, under some circumstances, be followed by no results so serious as to imperil life.

On the other hand there is always the likelihood that the foreign body may set up local infection. The experiments of Schüller showed that the ultimate effect of the introduction of particles of food through the windpipe into a rabbit's lungs was determined by their infectious or non-infectious nature.

When the substance was infective it produced fatal pneumonia, but when it was aseptic or practically aseptic it was either absorbed or remained inert or led, in time, to inflammatory changes of a low grade in the bronchi and pulmonary parenchyma.

The results of Schüller's experiments may be applied to human beings. It is possible for a foreign body to be tolerated in the lungs without serious damage, or it may lead to circumscribed cirrhosis and eventual bronchiectasis. In most instances bacteria gain access either because introduced along with the foreign body or because those already existing in the bronchi, as we know they normally do, penetrate beneath the endothelium (Hoffmann) or are carried by the reversed flow induced in the lymphatics into the part beyond the point of occlusion and occasion acute abscess or gangrene. In such an event the prognosis depends upon its early recognition and proper surgical management.

In other cases the destructive process may be a chronic one and lead to all the clinical evidences of pulmonary phthisis. The prognosis is also determined by the possibility of a superadded tuberculosis, an empyema, a pneumothorax or pneumonia in the opposite lung.

Although in the great majority of cases the foreign body may directly threaten life, still instances are on record in which the patient survived the accident for many years. Thus Gross is said to have observed a case in which life was prolonged to sixty years after the inhalation of a foreign body. Grains, grasses, straws and the like are considered especially dangerous and according to Hoffmann are particularly liable to introduce along with themselves actinomyces. See Actinomycosis of the Lungs.

Finally, the prognosis in the cases subjected to operation may be stated as follows: In the Practical Medicine Series of Year Books, vol. III Dec. 1902 The Eye, Ear, Nose and Throat, it is stated that combining the statistics of Durham, Gross, Weist and Coomes there are 2651 cases, and of these 1234 were operated upon with 964 recoveries. In 1417 cases no operation was performed with 1035 recoveries. "The percentage of recovery with operation brought down to date is 84.8." Judging from the context the operation referred to must have been bronchotomy.

Treatment.—The object of treatment is the removal of the foreign body as quickly and with as little likelihood of injuring the parts as possible. If the patient is seen immediately after the accident he should be aided in his attempt to dislodge the body by the act of coughing. A child may be seized by the ankles and held head downward while at the same time he is slapped vigorously on the back. An adult may be told to lie on his stomach across a bed and let his head and shoulders depend over the edge while he supports himself by his hands on the floor. In this posture he is to cough vigorously may at the same time be slapped between the shoulders.

Forcible inspirations tend to draw the foreign body still further into the chi and thus frustrate all attempts to dislodge it by the expiratory force oughing. Consequently the patient must be cautioned against taking a breath immediately before each act of coughing, but must be emphatically to cough without first drawing a deep breath. If the procedures just bed are intelligently carried out it may be possible to secure the expulsion of a foreign body that is round and smooth, as a bead or a bean, but bodies that are rough or have sharp corners or, like a head of grain, possess a prickly exterior are not likely to be so dislodged. On the contrary they are apt to be driven farther in with each inspiratory act. Hence the physician should endeavor to ascertain by inquiry the nature of the body that has been inhaled before resorting to the simple mechanical methods advised.

It is generally recognized by authors that a certain element of danger attends the expulsion of the body by the act of coughing, namely, the danger that it may be driven against the chink of the glottis, lodge there and cause fatal asphyxia. This is particularly the case in small children in whom the larynx is narrow. Nevertheless, as objects have been successfully expelled by the measures recommended, it is best to give them a trial.

The act of vomiting may sometimes aid in the expulsion of the offending body along with the contents of the stomach, and therefore an emetic may be tried in case of failure of the other means. Apomorphin hypodermically is the promptest and most efficient emetic and therefore should be selected. The dose is from $\frac{1}{10}$ to $\frac{1}{8}$ of a grain. The slight nausea which attends emesis serves to relax the tissues somewhat and, theoretically at least, may be assumed to loosen the foreign body in its position, while the secretion of mucus that attends the sickness may also favor the slipping of the object from its location in the bronchus.

Surgical measures are indicated whenever the foreign body cannot be expelled by the simple methods already described. They consist of bronchoscopy by Killian's upper direct method or of tracheotomy. The former procedure should be tried first and, in the hands of an expert laryngologist, may succeed even when the body is deeply situated in one of the main bronchi. Dr. E. Fletcher Ingalls has recently reported a successful removal of a foreign body by this means. In the case of children general anæsthesia is necessary to the successful introduction through the larynx and trachea of so long and wide a tube as is necessary. In adults, on the contrary, who have command of their nerves cocaine anæsthesia of the parts through which the bronchoscope is passed is generally sufficient.

It appears to be the consensus of opinion among surgeons and authors that tracheotomy should be resorted to as early as practicable after the failure of other methods. The statistics given by Durham, Weist, Gross and others, and quoted by various writers support the correctness of the advice for early resort to this operation, since the mortality of operated cases is less than in the unoperated ones.

The object of this operation is to facilitate the exploration of the bronchi and the removal of the body by suitable instruments. But an additional recommendation of tracheotomy lies in the fact that the body is sometimes coughed up through the wound. Whether either or both of the two measures is resorted to, care should be taken that the delicate mucous lining of the bronchi is not still further wounded by too prolonged and energetic attempts to grasp and extract the offending body. Failing these, bronchotomy may be resorted to with success in suitable cases.

Finally, should all attempts to locate and remove the foreign body prove

futile, the physician must await the possible development of a secon
monia and abscess or gangrene and then endeavor by physical sig
mine the exact location of the body with a view to having the abs
opened by a surgeon in the hope of discovering and removing th
cause, while at the same time treating the abscess.

Nevertheless, the attending physician should not be in too gre
advising surgical interference in cases of secondary pneumonia, si
pneumonia does not always terminate in abscess or gangrene. It
as shown by the history of many cases, for a foreign body to be endu
the lungs for many months or years and to be eventually expell
neously. The occurrence of grave septic symptoms in the course
aspiration-pheumonia, however, justifies the resort to lung surger
the foreign body can be located with sufficient certainty.

CHAPTER X

PERFORATION OF THE BRONCHI

ALLUSIONS have been so frequently made to this occurrence in preceding pages that a brief consideration of it is deemed advisable.

Ætiology.—Most cases are the ultimate result of compression, but perforation may be produced by a syphilitic or tuberculous ulcer. A foreign body in the bronchial tube is quite likely to cause perforation, either as a result of ulceration or, in rare instances, through injury inflicted by its sharp edges. It is said that Rokitansky met with an instance of fatal hæmorrhage in consequence of perforation of the innominate artery by a dart from a blowpipe which had lodged in the left main bronchus.

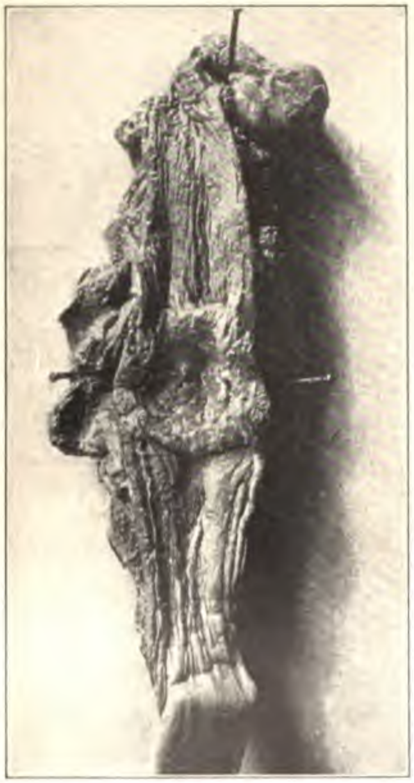


FIG. 13.—Carcinoma of œsophagus with ulceration.

Most frequently the causes are extrabronchial: aneurysm, cancer, an enlarged calcified lymph gland or the rupture of a so-called traction-diverticulum.

Pathology and Symptoms.—Prolonged compression of a bronchus leads to inflammatory swelling and ulceration of the mucous membrane, to absorption of the cartilaginous rings, and it may be to rupture of the bronchial artery. In this last event hæmoptysis is occasioned and may even prove severe. If the compressing force is an aneurysm it is possible, or indeed quite likely, that the sac may rupture with, of course, fatal hæmorrhage.

If, as is not very infrequent, the tube is perforated by a cancer of the œsophagus (Figs. 13 and 14), very striking and characteristic symptoms occur; namely, cough and expectoration of particles of food. Under such circumstances cough is very distressing, and if some of the fragments of food are aspirated into the deeper parts of the lung, aspiration-pneumonia and even gangrene may be produced.

Perforation by a calcified gland may not occasion appreciable symptoms, but if the gland be caseous acute broncho-pneumonia may ensue and prove the cause of death.

Perforation by a traction-diverticulum is brought about in the following manner. The rupture of the diverticulum causes an abscess in its immediate neighborhood in the mediastinum; the abscess then ultimately burrows its way until it at length opens into the same or another bronchus by one or several openings. Ziemssen and Zenker reported such a case in which the abscess cavity was situated in the anterior mediastinum and caused perforation of both main bronchi.

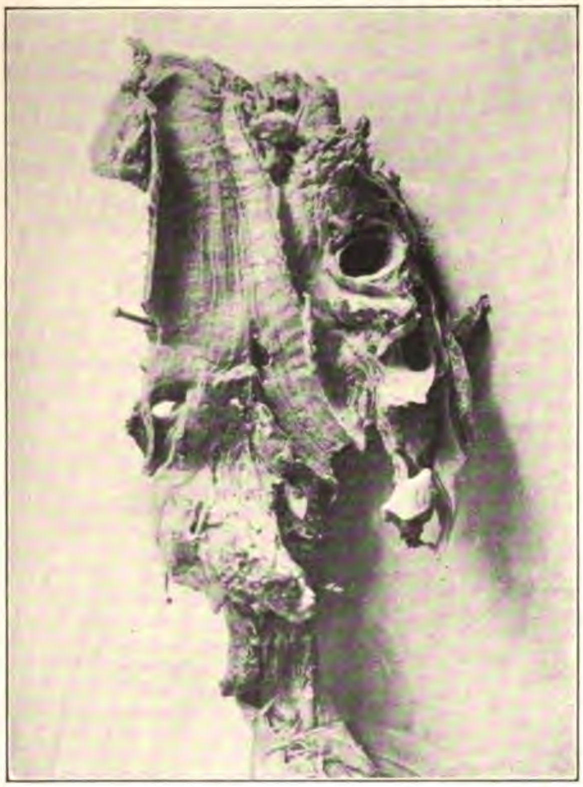


Fig. 14.—Bronchus perforated by carcinoma shown in Fig. 13.

The symptoms prior to the perforation are usually those of stenosis. The course is generally short, from nine to eleven days (Gerhardt) but may be as long as twenty-three days (Hoffmann).

The **diagnosis** may be easy, provided the physician is summoned immediately upon the occurrence of perforation, or in cases of aneurysm with resulting hæmoptysis and when food that has been swallowed is expelled by the act of coughing. The occurrence of hæmoptysis in a patient presenting symptoms of bronchial stenosis from other causes than aneurysm is suggestive of perforation. Gerhardt suggests that the diagnosis of a communication between the œsophagus and bronchus may be made by introducing an œsophageal sound and measuring the volume of air emitted from the sound (Hoffmann). If, after the sound has been passed beyond the point of communication, the amount of air which escapes becomes diminished, it indicates the transmission of air from the bronchus into the gullet and thus through the sound.

The **prognosis** is most grave. Death is likely to result from hæmoptysis, from the effects of the primary lesion, or from an aspiration-pneumonia. In exceptional cases the perforation may become closed and the symptoms may pass away.

Treatment is unavailing and must be confined to measures calculated to mitigate suffering.

SECTION II

DISEASES OF THE LUNGS

CHAPTER XI

DERANGEMENTS OF CIRCULATION

Pulmonary Anæmia.—Strictly speaking, anæmia of the lungs does not form a clinical entity; and even from the standpoint of pathology it cannot be said to exist independently, but to owe its presence to some local or general condition which is of far greater importance than the anæmia. It is present in chronic wasting diseases, as cancer, in the marasmus of old age, in extensive hæmorrhages and in the concentration of the blood seen in the last stage of Asiatic cholera, and in acute arsenical poisoning (Sticker). In these conditions, however, the pulmonary anæmia is so subordinate in its clinical manifestations to the general symptoms that aside from acceleration of respiration and some subjective sense of dyspnœa, it may not furnish any evidence of its existence.

There are two conditions, on the other hand, which produce anæmia of the lungs with very serious, even fatal results. These are pulmonary stenosis and embolism of the pulmonary artery. The former is, in the overwhelming majority of cases, a congenital affection, and almost invariably leads to pulmonary tuberculosis before the end of the third decade of life. The occurrence of phthisis in this form of cardiac disease is so frequent as to preclude the suggestion of their accidental association, and hence the anæmia of the lungs is considered responsible for the development of the tuberculous affection. It is not the result of diminished blood supply to the pulmonary system of vessels altogether or directly, but of impaired nutrition of the pulmonary parenchyma incident to lessened supply of blood to the aorta and hence to the bronchial arteries which are the nutrient arteries of the lungs.

Occlusion of the pulmonary artery or of one of its main branches is declared by symptoms whose gravity depends upon the completeness or incompleteness of the plugging. Sudden shutting-off of the blood supply to one and still more to both lungs is evinced by restlessness, rapid and labored breathing, cyanosis, tachycardia and feebleness of the pulse, in short, by all the signs of asphyxia ending in speedy death.

Certain pulmonary diseases may likewise be attended by general or circumscribed anæmia of these organs. Thus the paleness of the lungs seen *post mortem* in vesicular emphysema is due to this circulatory deficiency, and the anæmia is indirectly the cause of the respiratory embarrassment through the defective hæmatosis and consequent stimulation of the respiratory center.

Local anæmia attends compression of a lung by intrapleural accumulation of fluid, cirrhosis, etc., but unless so serious as to lead to marked congestion of the opposite lung it occasions no symptoms.

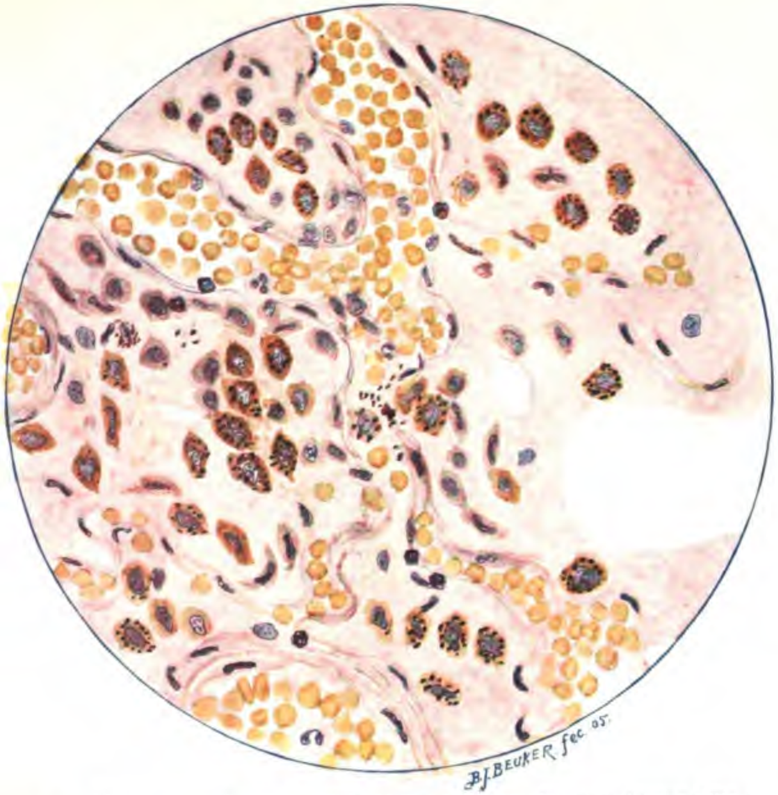
Pulmonary Hyperæmia.—Congestion of the lungs is divisible into two chief forms, *active* and *passive*. The former, also spoken of as acute and fluxionary, may be either circumscribed and local or diffuse and general, according to the condition occasioning the hyperæmia. When localized to one lung or a part of one lung it is usually an attendant upon or the result of some acute pulmonary or constitutional infection. Thus, for example, active hyperæmia of a lobe or of a limited portion of one or more lobes is present in the congestive stage of acute lobar pneumonia and in the incipiency of pulmonary tuberculosis. In such conditions it is difficult to decide whether mere congestion or inflammation exists, but it is likely that the fluxion is really inflammatory. This collateral fluxion, as it is called, also attends pleuritis and acute bronchitis when they interfere with the circulation in parts of one or both lungs.

Intense general congestion of the lungs is described in connection with great physical exertion, exposure to great heat and to cold, and, by some writers as Leuf, has been set down as the cause of death. The congestion resulting from excessive physical effort is by von Basch considered really a passive hyperæmia consequent upon failure of the left ventricle. Sticker, on the contrary, expresses the belief that in some cases in which the exertion is not attended by evidence of cardiac failure there is active hyperæmia of the lungs produced by the increased vigor of cardiac contractions. It seems not unreasonable to me that at first there may be an active pulmonary fluxion occasioned by the more rapid muscular contractions and the deepened respirations, but that when the undue exertion is prolonged there ensues an actual stasis within the pulmonary vessels, depending in its extent upon the degree of heart-fatigue. Osler's suggestion that in such fatal cases the coronary arteries may have been defective is very pertinent and merits careful consideration before the cause of death be attributed merely to acute pulmonary congestion.

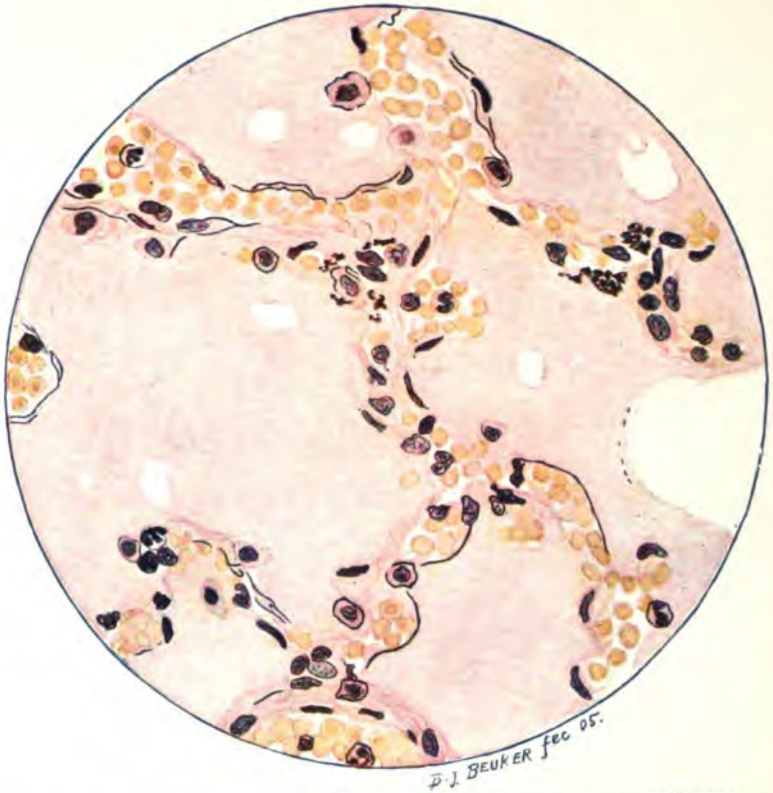
French writers, following in the footsteps of Woillez, attach much importance to active hyperæmia of the lungs and portray definite symptoms, as chill, elevation of temperature to 101° F., cough and dyspnœa, which strongly suggest an atypical or larval form of pneumonia. Likewise some of the cases reported in which a fatal pulmonary congestion occurred during gestation or as a result of great excitement seem to me to have been incompletely investigated in the light of modern pathology, or to have been misinterpreted. Intense hyperæmia may be the only definite condition discoverable at the autopsy, but in the absence of minute microscopic investigation of the state of the heart-muscle, it cannot be put down as the primary cause of death.

Passive hyperæmia occurs in conditions of the heart or lungs which hinder the outflow of blood from the pulmonary veins. Accordingly, it is

PLATE III



A.—Microscopic Section of Lung of Chronic Passive Hyperæmia. Note Large Number of Pigmented Cells Free in the Alveoli. (Stained with Hæmatoxylin and Eosin.)



B.—Microscopic Section of Lung of Acute Congestion and Œdema. Alveoli Filled with Coagulated Blood Plasma. (Stained with Hæmatoxylin and Eosin.)

found most often in connection with valvular lesions of the left heart, especially mitral stenosis. More or less congestion of the lungs is present also in cases of myocardial inadequacy from whatever cause. This form of hyperæmia is usually of slow development and long standing, but may supervene abruptly upon severe physical efforts which overpower the integrity of the heart-muscle. The condition is general in both lungs, as a rule, but may be confined to one lung or a portion of one lung when obstruction to pulmonary outflow is occasioned by pressure, as from aneurysm or tumor.

This form of passive congestion is known as *mechanical* and the changes induced in the pulmonary parenchyma constitute the so-called *brown induration* (Plate III). The lungs are large and found to cut and tear with difficulty. At first the cut surface is of a brownish-red color, whence the name, but exposure to the air turns the color to a bright red in consequence of oxidation of the hæmoglobin contained in the tissues. Microscopic examination shows distention of the capillaries with blood, and hyperplasia of the connective tissue elements; these changes account for the increased volume of the lungs. The alveolar walls are seen to be infiltrated with deeply pigmented cells while the interior of the alveoli contains deeply stained epithelial cells.

Animal experiments by von Basch and Grossmann have established the following facts with respect to changes in the lungs occasioned by pulmonary congestion when acutely induced, the effects being the opposite of those previously described as the result of pulmonary anæmia. Suddenly induced stasis within the pulmonic vessels causes swelling and increased volume of the lung with corresponding enlargement of the thorax, as shown by depression of the diaphragm and by mensuration. This distention of the lungs, which, when extreme, leads to rigidity or "lungenstarre" as it is termed by the Germans, hinders the excursion movements of the diaphragm and diminishes the respiratory expansion and contraction of the lungs, so that, although the breathing becomes labored, its mechanical effect is decreased rather than intensified, and the consequent stimulation of the respiratory center declares itself in the form of dyspnœa.

As remarked by Sticker, these results of animal experimentation are corroborated by clinical observation and afford satisfactory explanation of the symptoms witnessed in cases of pulmonary congestion. If swelling, rigidity and dyspnœa are the result of rapidly induced stasis, then we can understand the effect on respiration of the changes in size and consistency when long-standing hyperæmia has led to brown induration. In addition to dyspnœa there are more or less cough and expectoration in which latter are found deeply pigmented alveolar and eosinophile cells.

The effects on the lungs and respiration of pulmonary anæmia have been shown by experiments on animals, carried out by Grossmann in von Basch's laboratory. Suddenly induced anæmia of the lungs causes decrease in volume and relaxation of the lungs together with diminution in the size of the chest. Respiratory movements, on the part of the lungs, and the play of the diaphragm are increased, which greatly augments the mechanical effect of respiration upon the corresponding nerve-center, so that a period of apnœa at length follows the labored breathing. These effects are the exact opposite of what is observed in mechanical congestion of the lungs and explain the symptoms seen in cases of pulmonary embolism.

Hypostatic Congestion.—This is the term applied to that form of pulmonary congestion which is seen in persons confined for a long time in the recumbent position in consequence of fever, notably typhoid, or some other adynamic state. It may be observed in prolonged coma, injuries to the brain, and cerebral apoplexy when it is said to be more pronounced on, or even restricted to the hemiplegic side (Osler). This same author discovered it in two cases of death from morphine poisoning.

I recently attended a woman of seventy-one who, after lingering two weeks with left hemiplegia, the result of cerebral thrombosis, and with gangrene of the right foot from thrombosis of the anterior tibial artery, at length succumbed with symptoms indicative of hypostatic pneumonia. Without the development of cough and expectoration the posterior bases of both lungs, especially the right, exhibited dullness, and fine bubbling râles which obscured the breath-sounds. Slight pyrexia supervened and a few hours before death mounted to 101° F. This patient not only had remained in the dorsal decubitus for at least ten days because of the great pain in the gangrenous foot whenever an attempt was made to move her, but her heart was very feeble and her arteries were stiff, conditions which favored the occurrence both of the thrombosis in the leg and the hypostatic congestion of the lungs.

The foregoing case illustrates the causation of this form of pulmonary hyperæmia. The condition is termed hypostatic because supposed to be due to the force of gravity, the blood tending to sink to the most dependent portions of the lungs and to there stagnate. But inasmuch as individuals with healthy circulation may retain the dorsal decubitus for an indefinite time without showing signs of hypostatic congestion, it is apparent that some additional factor is at work. This factor is feebleness of the heart together with lessened vascular elasticity and is probably more influential than is posture. The congestion is therefore a passive one and in feeble patients is still further favored in its occurrence by the very sluggish superficial respiration incident to their weakness and prolonged muscular inaction. The congestion of the lung bases seen in abdominal affections, ascites, meteorism and tumors, which occasion upward pressure, is probably due largely to deficient respiratory movements.

Examination of hypostatically congested lungs *post mortem* shows the posterior portions of a dark red color, heavy and more or less airless, even to the extent of sinking in water. That this condition is due to engorgement with blood and serum is shown by the fact that the cut surface often drips a bloody fluid. This condition so closely resembles the cut surface of the spleen as to have received from old pathologists the term splenization.

Hypostatic congestion does not present a definite or characteristic clinical picture. In fact, in a case in which it is likely to occur, e. g., severe typhoid fever, it may not anywise modify the symptoms of the original disease. It must be sought for consequently by careful and repeated physical exploration of the lungs.

The diagnosis rests on the detection in suitable cases of moderate dullness at the base of one or both lungs behind, together with increased resistance, feeble bronchial breathing and fine moist râles. In very feeble patients without fever due to the primary affection, slight elevation of body temperature may attend or supervene upon the development of signs of hypostatic congestion. It is in such cases that the term hypostatic pneumonia is especially applicable. I suppose it is very possible for a terminal infection to take place in such cases.

Pulmonary Œdema.—This condition is frequently encountered in cases of cardiac disease, either as a terminal event or as a transient condition of very grave danger which, however, yields to appropriate treatment. The lungs are not only engorged with blood but drip with serum from their cut surface and sink in water, since the air-cells are filled with transuded fluid (Plate III). In such fatal cases the œdema is general in both lungs, but a collateral œdema not uncommonly attends pneumonic infection or severe pulmonary inflammations and is then limited to the tissue adjacent to the inflamed area.

The mode of occurrence of this condition has attracted much attention and received both speculative and experimental study, notably by Cohnheim and Welch. Although mechanical pulmonary congestion may reach so extreme a degree and persist for so long a time that the tissue becomes more or less œdematous, still pulmonary œdema is something more than mere stasis. It appears reasonably well established that the condition is produced by a suddenly developed and pronounced disproportion between the strength of the two ventricles. In consequence of potential weakness on the part of the left ventricle this chamber becomes suddenly incompetent while the right ventricle goes on discharging its contents into the pulmonary artery with its accustomed energy. This leads to an abrupt and overwhelming engorgement of the pulmonary vessels with resulting transudation of serum into the tissues and alveoli. Our knowledge of the causation of dropsy in other parts of the body makes it not unlikely that still another factor favors this osmosis of fluid within the lungs and this is a nutritional disorder on the part of the capillary walls. This surmise does not seem to me at variance with the fact that pulmonary œdema is a frequent attendant upon the death agony. Moreover, if it were merely the expression of a suddenly induced stasis, then it ought to occur far more often than it does in cases of cardiac disease. I have known of its having occurred twice in the same female with mitral regurgitation, in both instances as a result of physical exertion which in other individuals with the same valve lesion would have occasioned no more serious symptoms than dyspnœa and cough.

In a considerable number of recorded cases thoracentesis has been attended or succeeded by *acute pulmonary œdema*. Either during the operation or an hour or two thereafter the patient begins to cough and to expectorate large amounts of frothy often viscid, transparent or yellowish-green serum which may flow into the air-tubes more rapidly than it can be expectorated and under such circumstances may be a cause of death by asphyxia. This serous fluid is coagulated into an almost solid mass by heat or the addition of a few drops of nitric acid, is of a specific gravity of about 1.020 (Gee) and is rich in albumin together with mucin.

That such a mishap does not follow every case of paracentesis thoracis indicates that when it does occur there is some complication, as adhesions, or a faulty state of the blood-vessels or some defect in the technique of the operation, as a too rapid withdrawal of the exudate. In a case reported by Magenau the œdema of the lungs was attributed to chronic mediastinal adhesions aided by trophic disturbance of the pulmonary capillaries.

The symptoms of pulmonary œdema are a subjective feeling of intense oppression within the chest and dyspnœa which are speedily accompanied by cough and the expectoration of abundant frothy or in some instances bloody serum. Objectively the chest is perceived to emit a multitude of fine crackling râles while pulmonary resonance is but slightly if at all impaired. In a case I vividly recall having been presented in von Ziemssen's clinic at Munich the minute crackling sounds were audible to us students sitting at a distance on the benches and strongly resembled the sound of salt thrown upon a hot stove. This state may persist for hours or may speedily cause the death of the patient.

Pulmonary Embolism and Infarct.—Embolism of a pulmonary artery is a not very infrequent occurrence which may or may not carry in its train consequences of the gravest possible moment. If the embolus be of infective nature and enter a small branch a localized abscess or gangrene may be the consequence (see chapter on Pulmonary Abscess and Gangrene). If, instead, the embolus be of a benign or noninfective character the results depend on the size of the artery concerned. If this be small there is a circumscribed extravasation of blood into the tissues of the area supplied by the occluded vessel. To this condition, therefore, is applied the term hæmorrhagic infarct; and, as is well known, the lungs furnish one of the most frequent seats of such an infarct.

Laennec appears to have first recognized the existence of such localized hæmorrhages and he gave to them the name of pulmonary apoplexy, but he did not appreciate the importance of occlusion of an artery as the prime essential of the local hæmorrhage. This was first done by Bouillaud in 1826. Nevertheless it was the immortal pathologist Virchow who first recognized pulmonary infarcts to be of embolic origin and not due to thrombosis. He was compelled to assume the cause of the infarct to lie in embolism since he was not able to demonstrate it conclusively by experimental occlusion of the arteries with minute particles of rubber.

Nevertheless subsequent experiments by Cohnheim, Litten, Fujinami and others proved that if some other substance as paraffin was used, it was capable of producing infarcts and hence that Virchow's experiments failed because the obstruction to the arteries was not complete. Accordingly, these experiments determined that an absolute plugging of the vessel is essential to the formation of a hæmorrhagic infarct. There are other conditions, however, which are necessary and which as yet can scarcely be said to be conclusively established. The area occupied by a hæmorrhagic infarct is wedge-shaped with its apex toward the hilus of the organ (in this case the lung) and its broad base toward the periphery. Cohnheim's which obtained for a considerable time was, that as the plugged an end artery a back flow took place through the capillaries into the ischæmic territory with subsequent extravasation of blo tissues.

Without entering into the many experiments and discussions taken place to prove or disprove the correctness of Cohnheim's the be stated that this explanation of the pathogenesis of pulmonary now known to be incorrect. Kuettner and Litten showed that might occur when both artery and vein were tied, but that it di place if all the vessels were cut which united the circulation of the to that of the aortic system, namely, the bronchial arteries and the œsophagus, trachea, pericardium, mediastinum and diaphrag thus shown that the establishment of a collateral circulation is n an infarct is to follow the embolic occlusion of a pulmonary arteri

The explanation of the pathogenesis of hæmorrhagic infarcts i is rendered still further obscure by the consideration that the co pears to take place sometimes in the absence of a discoverable embol theless it would seem probable that in such instances there reall embolic plugging of the vessel but that the plug is not discovered tigators all recognize the extreme difficulty of discovering an embolis instance. Fujinami therefore expresses the belief that the absence bolic occlusion is apparent rather than real. Fraenkel appears a this view since he says that from the standpoint of the clinician ha pulmonary infarcts are most often seen in precisely that class of cas cardiopaths, in which emboli would be most likely to occur.

Again it has been shown that it is possible for a pulmonary become plugged without the establishment of a hæmorrhagic infar there must be some additional factor in the pathogenesis of this This additional element appears, from the painstaking investi Willgerodt, to consist in stasis in the pulmonary system (Fraenkel) ingly it is either when pulmonary hyperæmia has reached an extr as in mitral stenosis or in grave cardiac asthenia from any cause, t rhagic pulmonary infarcts are most likely to occur. The stasis in nary capillaries and veins interferes with the establishment of circulation in the anæmic area sufficient to maintain the nutrition since the collateral circulation is not strong enough to withstan flow from the veins. The capillaries of the area undergo increasing until at length extravasation of blood ensues and the hæmorrhagic the ultimate result.

The origin of the emboli is various. In cases of heart diseas or plugs, as the case may be, have their origin in the right heart. quence of stagnation of the blood in the auricle or at the apex or l trabeculæ carneæ of the ventricle, coagula are formed from which of differing size may be broken off and thence be swept by the bl into some portion of the pulmonary system. Accordingly it is i extreme mitral stenosis that pulmonary infarcts are most likely to

Emboli may, however, arise from thrombosis within the veins of various parts of the body, e. g., the femoral vein, the venous sinuses of the brain, the veins of the uterus after parturition, the veins of the prostate, of the liver (Fig. 15) etc. In fact, wherever thrombosis is occasioned, there may be produced all the conditions essential to the formation of an embolus, especially if it be an infective process that occasions the thrombosis, since the clot formed under such circumstances is very easily broken down. Therefore, the nature of the primary process influences the character of the changes that ensue in the infarcted area.

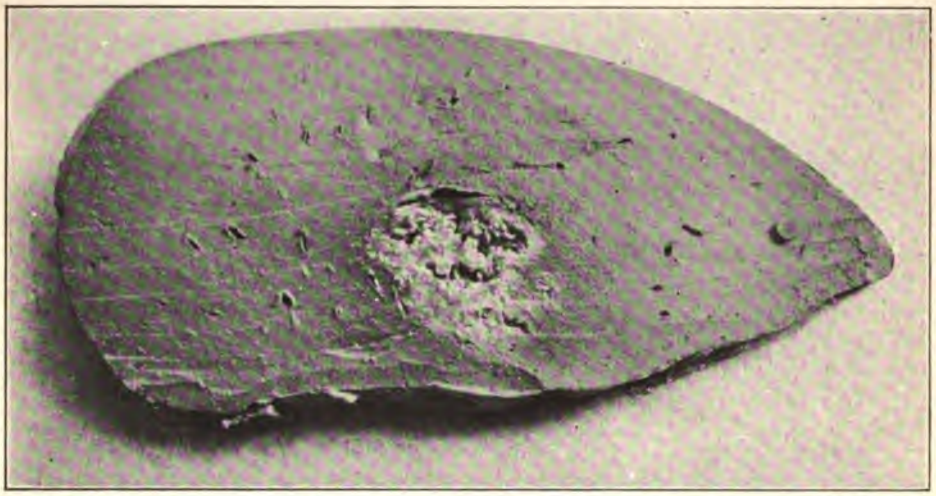


FIG. 15.—Abscess of liver from which arose emboli lodging in lung shown in Fig. 16.

Emboli of other sources may also gain access to the lungs. Thus thrombotic masses in cases of malignant endocarditis affecting the valves of the right heart may become detached and be carried into some branch of the pulmonary artery with very disastrous consequences. Fat emboli also occur in bone fractures, especially when the spongy bones are crushed. In such instances fragments of the bone marrow may be carried into the vessels of the pulmonary system and form a source of great dread to the surgeon. Air-emboli and emboli made up of micro-organisms or of tissue cells may also be carried by the blood-stream into distant parts, among these the lungs. They do not, however, constitute as great a source of danger as do those previously mentioned.

The size and number of the emboli differ enormously in different cases. As may be deduced from such variations the consequent changes and effects vary within wide limits. If the plug be of sufficient size to occlude the main stem of the pulmonary artery, death is the speedy result; when, on the other hand, a branch of considerable caliber is plugged, the infarct may occupy the greater portion of a lobe and then furnishes a marked example of pulmonary apoplexy. In the majority of instances the emboli are small and of considerable number and lodge in the lower lobe along both its posterior and inferior border.

It is an interesting circumstance that infarcts are more common in the right than in the left lung, a fact which, by Gerhardt, is attributed to the greater force of the blood-stream in the right branch of the pulmonary artery. This explanation seems borne out by the observation that when the current of blood passing to the right lung is diminished in force in consequence of some condition, e. g., collapse secondary to a pleuritic exudate, fibrosis and atrophy, the emboli become more numerous in the left lung. Penzoldt, moreover, has shown that such is also the case when, on account of prolonged right lateral decubitus, the left lung is required to perform increased respiratory play.

The shape of a pulmonary infarct is that of a wedge having its apex directed toward the center or hilus of the organ. If it is situated at the periphery the pleural membrane overlying the infarct is somewhat cloudy while the area itself presents a bluish appearance and is slightly raised above the surface of the neighboring part. The cut section is smooth or slightly granular and may permit a little blood to escape. At its apex is usually discoverable by its whitish appearance a small plug which is the embolus. Should the artery not be completely blocked at first by the plug this is speedily produced through the formation of a thrombus roundabout.

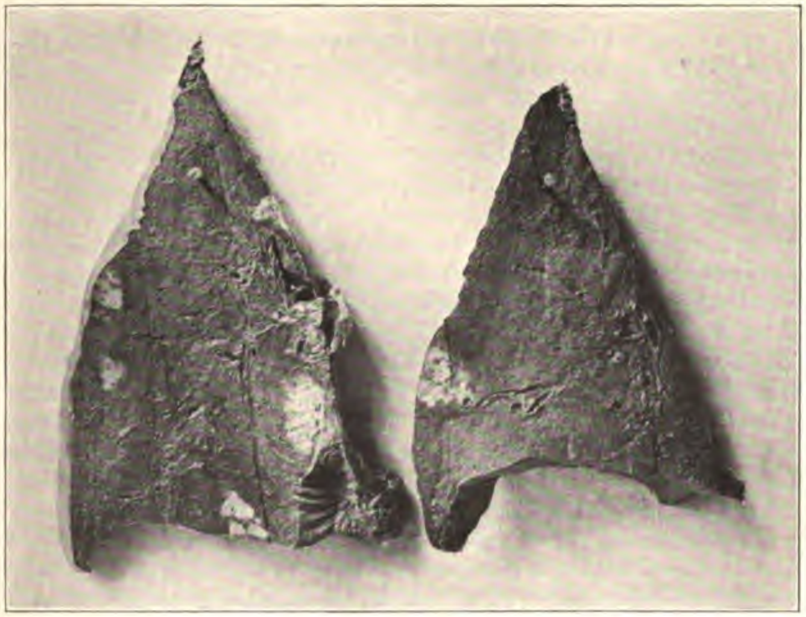


Fig. 16.—Septic infarcts of lung secondary to liver abscess shown in Fig. 15.

If the embolus be infected the inflammation resulting is very apt to result in necrosis (Fig. 16) and abscess or gangrene. But if the effect be confined to a mechanical occlusion merely, with hæmorrhage by diapedesis the extravasated blood may in time undergo absorption. The connective tissue of the area takes on a hyperplasia and the infarcted district becomes cicatrized. This newly formed tissue at length contracts and in time the site of the infarct is shown by a depressed, whitish area having the characteristic wedge-shaped outline.

The symptoms of pulmonary embolism depend upon the size of the artery that is plugged. If it be the main stem of the pulmonary artery or one of its two chief subdivisions that is occluded the result is apt to be death, the fatal issue being ushered in by most distressing symptoms.

The patient, for example, a woman who has been recently confined, experiences a feeling of profound oppression and anxiety or a sense of impending dissolution, springs up in bed and gasps for breath while the countenance assumes an ashen hue which may be speedily succeeded by cyanosis of the lips and cheeks. The respiration becomes extremely rapid and the pulse is greatly accelerated and almost or quite imperceptible. These symptoms persist for a brief time, minutes to hours, and death occurs. In some instances a convulsive seizure takes place and the patient dies unconscious without portraying the dyspnœa and suffering shown in other cases.

The cause of death is variously attributed to paralysis of the heart from sudden interruption of its blood supply or to cerebral anæmia in consequence of the interference with the flow of blood to the left ventricle and hence to the carotids. It is particularly in the cases characterized by convulsions and unconsciousness that anæmia of the brain is thought to be the immediate cause of death.

If physical examination of the chest be made in these terrible cases the area of præcordial dullness is found increased to the right, in consequence of acute dilatation of the right ventricle, this dilatation being either a manifestation of the abnormal resistance in the pulmonary circuit or the result of deficient blood supply to its myocardium (Fraenkel). The lungs may or may not evince any recognizable changes on percussion and auscultation. Thus the occlusion of a large branch of the pulmonary artery may be declared by signs similar to those in cases of extensive atelectasis, namely, diminution of fremitus and dullness at one or the other base, with loud bronchial breathing or enfeebled or indeterminate breath-sounds over the respective area. Ordinarily however death occurs too soon for the attending physician to make a careful chest examination, or such an examination proves entirely negative.

If demonstrable findings are present they make their appearance only after time has been given for the effects of the embolism to declare themselves. Thus, in a case of embolic pneumonia following occlusion of the right pulmonary artery or one of its large subdivisions, Landgraf is said to have observed feebleness of the breath-sounds over the affected lung, and in front, dullness down to the upper border of the liver. Over this area breathing was diminished. The diaphragm assumed a somewhat elevated position and the respective half of the thorax showed retardation in its respiratory movements.

Litten has called attention to the occurrence of a systolic murmur and thrill in partial plugging of the main artery or one of its great divisions, which murmur will have its location over the seat of the artery or its bifurcation,

namely, at the left of the sternum at the level of the second or
cartilage, and in the interscapular region opposite the third dorsal
also reported in one instance a doubling of the apex beat which h
to delayed systole of the right ventricle, in consequence of its bein
as compared with the relatively empty left ventricle (Fraenkel).

The symptoms of a hæmorrhagic infarct are quite different fr
going and depend upon the circumstance that the embolus is of rela
dimension. If it be located beneath the pleura the patient usually
a more or less sharp and sudden pain in the side of the chest. In so
there may be a rigor which in time is supposed to correspond with t
of the embolus. A variable length of time thereafter (in one o
cases eight and one half and in another twenty-four hours), but us
a comparatively short time, occurs the expectoration of bright r
of mucus tinged more or less deeply with blood. In amount this bl
may vary from a few mouthfuls to a considerable number of ounce
persist for days or even weeks. In most cases, if repeated inf
occur, the hæmoptysis gradually ceases or the expectoration ma
assume the appearance of dark nearly black blood.

The temperature of the body may or may not be elevated; bu
of a febrile character it is not high nor of long duration, except
in which abscess or gangrene result. According to Gerhardt a
the temperature rarely reaches 104° F. but remains below 102.2

Should the embolus contain pyogenic or saprophytic organis
and general consequences are those of embolic abscess or gang
single or multiple. The temperature becomes pronouncedly sep
sputum, at first hæmorrhagic, at length displays the characters
the local process; that is, it contains hæmatoidin crystals and sh
monary tissue or is greenish-black and fœtid. The consequent i
the bronchial mucous membrane excites frequent and severe c
strength and nutrition fail rapidly and death is very apt to res

In any case of superficially located embolism there is apt to
pleuritis which may result in an exudate of greater or less amou
of recognition; or the pleurisy may, in infective cases, display th
of a putrid pleuritis. According to Gerhardt, the occurrenc
pleurisies in the course of cardiac disease may generally be asc
farcts. And what is very important of recollection, the pleuris
date may be the only indication of the infarct, there being no e
of blood to announce its occurrence.

Fat embolism within the lungs may not occasion definite or
symptomatology when their number is not great. When, howev
numerous or of large size there may be the findings of acute pulmo
preceding the fatal issue. The occurrence of symptoms refer
respiratory system in any case of crushing injuries to the b
always arouse the suspicion of fat embolism in the lungs. Th
sudden death after such a trauma may, with reasonable assuranc
uted to such a cause.

The diagnosis of embolism of the pulmonary artery or of one branches must be based on history and symptoms rather than on physical findings at the bedside. Given some condition in the course of which the dislodgment of a clot is possible, e. g., during the puerperium or a thrombosis of the femoral vein, etc., and the sudden intervention of urgent dyspnœa together with signs of failing heart, one may with good reason attribute the phenomena to the catastrophe in question, since probabilities are in favor of embolism. There is scarcely any other condition so likely to occur. Many times death comes too quickly to admit of positive diagnosis; but should some hours elapse after the onset of urgent symptoms, careful examination of the lungs should be made and, should signs denoting the exclusion of air from a considerable area at one base be detected, the diagnosis may be considered established.

In cases of pulmonary infarcts there are several features which enable one to arrive at a diagnosis with certainty in most instances. A sudden pain in one side of the chest with perhaps a chill; the subsequent expectoration of pure blood or bloody mucus; the development of a febrile temperature in some, though not all, cases; and the existence of serious heart weakness or of an extreme degree of mitral narrowing; these form a complexus of symptoms which can scarcely belong to any other condition than embolic plugging of one or more arterioles within the lungs. If, in addition, there be discovered an area of dullness with bronchial or diminished breath-sounds at the posterior or lateral base of one lung, the correct interpretation of the case is not difficult.

When, however, bloody expectoration and pain in the lung do not occur the diagnosis may be impossible. Infarct may be inferred however in cases of cardiac disease, especially in acute endocarditis, when, without apparent cause, an acute pleurisy develops (Gerhardt). The same holds true with respect to the sudden onset of signs of pulmonary œdema soon after fractures or crushing injuries, since it points with much certainty to fat embolisms within the lungs.

The prognosis is always grave, and in cases of total plugging of the main trunk of the pulmonary artery is hopeless. Incomplete occlusion of the large branch may admit of eventual recovery to a tolerable state of health, since it is possible for compensatory increase of blood-pressure in the sound parts of the pulmonary system to maintain the supply of blood needed for the preservation of life. In the vast majority of cases, nevertheless, death is the consequence.

Hæmorrhagic infarcts do not in themselves cause death unless they be very extensive or very numerous. In most cases it is the underlying disease, e. g., mitral constriction, which destroys life. They furnish a bad prognosis, however, because pointing to a condition which in itself is hopeless or which contains the possibility for repetitions of the embolism.

Treatment of Circulatory Derangements.—**Pulmonary anæmia** is amenable to therapeutic measures only in so far as they may favorably affect the underlying condition. In the majority of instances stimulants are indicated and these are to be conjoined to such other measures as will increase cardiac and general strength.

Pulmonary Hyperæmia.—The treatment indicated for this condition is appropriate to either form, the active or the passive. Absolute physical rest is of the first importance and, in the slighter cases, will usually suffice to bring about an equilibrium of the circulation. Should acute dilatation of the right ventricle and marked signs of stasis in the systemic veins be present and the state of the pulse portend danger to life from paralytic overdistention of the cardiac chambers, recourse should be had to prompt bloodletting. A vein in the arm may be opened without needless delay and twenty to thirty ounces may be allowed to flow.

Should the stagnation be such that relief is not afforded, then one may without fear resort to aspiration of the right auricle. An aspirating needle may be thrust into this chamber close to the right border of the sternum, care being exercised to avoid wounding the internal mammary artery which passes from $\frac{1}{3}$ to $\frac{1}{2}$ of an inch from the edge of the bone. This procedure is not dangerous since the myocardium endures puncture with a fine needle without subsequent bleeding from the wound. Since, however, this operation is advisable only in cases of grave danger to life, the risks of the puncture are not to be weighed against the disastrous consequences of delay or a too timid treatment.

The withdrawal of blood by either means may be followed by the administration of diffusible stimulants by mouth or under the skin. Of these the most efficient are those recommended in the treatment of the threatening heart failure of acute pneumonia, and for the enumeration of these the reader is referred to that chapter.

Other remedies of service in congestion of the lungs are cathartics, digitalis and morphine. The first are beneficial by causing a revulsion of blood from the pulmonary vessels to those of the abdomen and in cases of chronic heart disease are indispensable. Digitalis finds its indication in all forms of cardiac asthenia and particularly in the chronic valvular lesions underlying most cases of passive pulmonary hyperæmia. Morphine is a most valuable agent in these cases since it is a powerful stimulant in small doses hypodermically and also acts as a sedative to the nervous system. It possesses the merit also of being a sedative to cough and to this end may prove very necessary.

The application of cold compresses to the skin of the chest in the manner recommended for hæmoptysis is highly indorsed by certain French writers. The reader is also advised to read what is said in the treatment of pulmonary hæmorrhage concerning the application of a hot-water bottle to the cervical and upper dorsal regions of the spine. Patients likely to develop hypostatic congestion must not be allowed to remain too long in the same position, and may be given hot drinks and other harmless stimulants.

Pulmonary œdema calls for prompt and energetic cardiac stimulation. A hypodermic of morphine is generally resorted to by most practitioners, but by Huchard and others is not considered safe. One fiftieth or even one twenty-fifth of atropine injected under the skin is far more efficient and is quite rational, since it promptly bleeds the patient into his own peripheral vessels and is not dangerous even in the latter dose to an adult. I have known the threatening pulmonary œdema of acute pneumonia to be arrested as if by magic by a hypodermic of $\frac{1}{80}$ of atropine. The abstraction of blood is not rational in this condition, since what is needed is not to relieve stasis but to arouse the flagging energy of the left ventricle. The hypodermic administration of adrenalin is considered dangerous and is certainly less efficient than the injection of atropine.

T. S. Southworth who observed an acute pulmonary œdema in an infant of thirteen months which but shortly before had had pneumonia reports prompt relief by the inversion of the tiny sufferer. At the same time while the baby was held head down the chest was stroked over the large bronchi from the base toward the neck. A small amount of bloody serum ran from the child's mouth and nose, and when at length the infant was returned to its bed with its feet elevated and surrounded by hot bottles, its relief was very apparent. Southworth believes this procedure saved the patient's life.

Acute pulmonary œdema following the aspiration of pleuritic effusions cannot always be prevented, but an attempt to do so should be made by the slow and careful withdrawal of the exudate. Should the œdema occur in spite of such precautions, then efforts may be directed to the relief of the œdema, in the form of digitalis, stimulants, cathartics, diuretics, etc.

Pulmonary Embolism and Infarct.—The complete plugging of the pulmonary artery or of one of its main divisions cannot be said to be amenable to treatment. In many instances death comes too soon to afford time for the institution of therapeutic measures. When, on the other hand, some hours elapse and the physician is warranted in the inference that one of the large branches and not the trunk of the vessel is the seat of embolism, then recourse should be had to such measures as appear indicated. These in the bulk of cases are stimulants and morphine. Camphor, musk, ammonia, ether and brandy may any or all of them be administered hypodermically and without delay. Morphine given in the same manner is certainly indicated, for it will at least calm the patient and obtund the sense of dyspnœa. If the state of the sufferer is one of collapse, the physician should also order the application to the extremities of hot bottles, sinapisms and the administration of hot stimulating drinks, to assist the stimulants previously mentioned, in the restoration of circulation to the superficial parts.

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CHAPTER XII

PULMONARY HÆMORRHAGE

HÆMOPTYSIS

Ætiology.—In a strict sense bleeding from the lungs includes all forms of hæmorrhage from bronchial or pulmonary vessels whether the result of *external injury* as gunshot or stab wounds, or of *pathological conditions* residing within the organism. The term also embraces cases in which blood is effused into the pleural cavity or remains concealed within the pulmonary parenchyma as well as cases in which it escapes through the mouth (hæmoptysis).

External Injuries.—Bleeding from the lungs in consequence of external injury belongs to the province of surgery and will not be considered in this work. Hæmorrhage into the pleural cavity may likewise be regarded as a surgical affection, and will not here be discussed at length. It may take place in consequence of paracentesis thoracis through the wounding of an intercostal artery or of penetration of the lung by the needle, but it would appear to be possible without the intervention of external agency. This so-called spontaneous hæmatoma is in most instances due to tuberculosis of the lungs (Dieulafoy, Moutard-Martin, Mesnil) and runs a favorable course. But hæmothorax may according to Sticker owe its existence to influenza, typhus fever, cirrhosis of the liver, Bright's disease, scurvy, malaria and cancer of the lung.

In the midst of apparently good health and without signs of local disease in the lung there may develop dyspnœa and other symptoms of exudate within the pleural cavity which may amount to two or even three liters. The nature of the effusion is only ascertained by exploratory puncture. The urgency of the symptoms may necessitate aspiration, or, after a couple of weeks, the fluid may be spontaneously absorbed. The hæmorrhage is said not to evince a tendency to recurrence.

Sticker briefly narrates Mesnil's case of a seemingly healthy farrier of thirty-two who, after unusually strenuous work, was seized with a chill and manifested evidence of a left-sided pleuritic effusion. On the eighth day thereafter his dyspnœa increased suddenly and there were frequent chills but without febrile temperature. Three days subsequently the difficulty in respiration necessitated thoracentesis and two and one half liters of blood were withdrawn. Recovery rapidly ensued without the patient's displaying temperature at any time. The tuberculous nature of the bloody effusion was proven by the inoculation of guinea-pigs.

Sticker also cites Gendrin's case in which the diagnosis of hæmatopneumothorax was made but not substantiated by puncture. A twenty-three-year-old servant girl became greatly excited during her catamenia which resulted in prompt suppression of the flow. The next morning she experienced dyspnœa and chest-pain and two days later cough and copious hæmoptysis developed which attack was repeated next day but with still greater violence of the cough. During this paroxysm she was seized with severe pain in the left side and was removed to the hospital where the morning following were discovered signs of pneumothorax together with fluid. There was considerable elevation of temperature which began to subside on the sixth day. On the ninth day of the illness cough disappeared and on the thirtieth the dullness began to show signs of clearing up with returning breath-sounds of a vesicular quality, and on the fifty-sixth day the patient was discharged well but with signs of retraction of the affected side. Gendrin classes this case as an instance of vicarious menstruation, but Sticker prefers to consider it one of pulmonary tuberculosis believing that such accidents do not occur in lungs which are previously perfectly healthy.

Hæmorrhage from the lungs may result from crushing injuries to the chest-wall, violent blows, falling from a height, etc. The bleeding may be internal, as into the pleural cavity, when it is declared by increasing pallor and weakness that may go on into a state of collapse, or it may be external as well as internal. In the latter event blood enters the air-tubes from the lacerated lung and is expectorated.

That hæmorrhage into the pleural cavity may result from laceration of the lung by violent cough is shown by Bonin's case, quoted by Sticker, of a certain Dr. Fortassin who, after having suffered for a short time from cough and oppression of the chest, was found dead in his bed one morning, although the evening previous he had partaken of a hearty supper and retired in usual health. The *post-mortem* examination disclosed the right pleural cavity filled with partially coagulated blood which had evidently escaped from several lacerations in the upper anterior surface of the lung. The pulmonary tissue and air-tubes were likewise infiltrated and occluded by blood. Sticker very justly remarks that the examination of the lung was not complete, but that nevertheless the case may be regarded as a proof of the fact that laceration of the lung (presumably at some diseased point) can be occasioned by violent tussal efforts.

In cases of traumatic pulmonary hæmorrhage there may be, in addition to the symptoms of shock and even collapse, signs of the collection of fluid in the pleural cavity; or the fact of injury to pulmonary tissue may be shown by the escape of blood from the mouth by the act of coughing.

The frequency of hæmoptysis in these cases is, according to statistics, very variable, but on the whole cannot be said to be great. Of 401 instances of contusions of the thorax without external wound collected from the military reports of the Franco-Prussian War of 1870–71, hæmoptysis was observed but forty-three times, or 8.7 per cent, whereas Mossakowski witnessed bloody sputum forty times in forty-three cases.

This symptom does not always appear immediately or soon after the

trauma. Thus Stricker who saw traumatic pulmonary hæmorrha
four times among soldiers, reports hæmoptysis directly following
in three instances, within the next twenty-four hours in three ca
seven others on the third, fifth, seventh and eighth days, and in o
not until after the lapse of two months.

It is also worthy of note as bearing on the influence of previ
on the occurrence of hæmoptysis, even after trauma, that Stricke
to determine the positive existence of pulmonary tuberculosis i
his cases, and its probable presence in six. Furthermore, the s
question took place directly or within a few hours following
in those soldiers who were free from tuberculous disease, whereas
were either unmistakably or probably tuberculous did not bleed
the lapse of days.

Pathological Conditions.—A great variety of diseased state
rise to external pulmonary hæmorrhage or hæmoptysis. Most of
sist of definite lesions which can be demonstrated *post mortem* i
cally, but, as will be seen in discussing hæmoptysis as a manif
vicarious menstruation, it is not always possible to discover struc
as a cause of the bleeding even when there can be no doubt of
having come from the lungs.

(1) **Pulmonary Tuberculosis.**—This is so frequently the
process underlying hæmoptysis that, when this latter occurs,
suggests to physician and laity the possibility of pulmonary t
even though other symptoms, as cough and loss of weight, have
or have been too insignificant to attract attention. Several q
interest and importance arise in connection with this symptom,
stage of the disease in which it most frequently occurs or is likel
(*b*) the source of the hæmorrhage, (*c*) its effect on the progn
primary or underlying tuberculosis. These queries will now be
in turn and satisfactorily answered so far as is possible.

(*a*) *The stage of the pulmonary tuberculosis* in which hæmopty
often observed and hence in which it may be expected to occur, i
given. Some writers, among the many consulted on this point
precise statement concerning its relative frequency in the various
like Osler, merely mention hæmoptysis as frequently being an initia
Others give statistics bearing on this point. Thus Laennec is sa
to have reported hæmoptysis as a first symptom in 36 per cent an
occurred within the first month in 50 per cent. It is misleading t
blood-spitting as the initial symptom in any case, for in all
some other has preceded it but has been overlooked. It is better
to study this symptom with reference to the early or late stage of
culosis. Cornet states that hæmoptysis may occur at any time
often in the early stage and most authors coincide in this opini

Thus Wilson Fox is authority for the statement that in the first
Hospital Report, hæmoptysis is put down as having occurred befor
i. e., early in the disease, in 73 per cent of males and 72 per cent
He remarks later on, however, that these figures are opposed to the

of most observers who find hæmorrhage more frequent in the stage of softening and excavation. Nevertheless, Fox cites Williams as having recorded profuse hæmoptysis in 66 per cent of early phthisis, and Walshe as stating that "in upward of half of the cases of notable hæmorrhage, this occurs or has occurred as a first symptom." Samuel West, on the other hand, does not so far as I can find make any definite statement on this point.

Sticker expresses the opinion that an initial hæmoptysis takes place in about a third of all cases. Fraenkel contents himself with the statement that blood-spitting occurs in a certain proportion of cases as the first symptom on the part of the respiratory apparatus, which impels the patient to seek medical aid, but has usually been preceded by other symptoms.

C. J. B. Williams says, with reference to the first Brompton Hospital Report, that "hæmoptysis is more frequent (as three to one) before softening than after that process has taken place." His own cases support this contention, since out of 283 cases of hæmoptysis taken from his private practice 187 were in the first stage, sixty-five in the second and thirty-one in the third stage. Walshe gives hæmoptysis in the first stage as occurring in 71.79 per cent. On the other hand, Reginald Thompson's cases are acknowledged by Williams to make for the greater frequency of hæmoptysis after softening has begun. From the foregoing it would appear that this symptom is more frequent in the early than in the late stage of tuberculosis.

As regards the *relative frequency* of its occurrence throughout the course of the disease figures agree fairly well: Louis 60 per cent of all cases, Gerhardt 80 per cent. As respects my personal experience I see this symptom most often in the early stage, which is not strange since, in the majority of tuberculous cases I meet with my services are solicited for the purpose of diagnosis.

(*b*) *The Source of the Hæmorrhage.*—Another point of interest raised by the occurrence of this symptom pertains to the source of the blood, whether from a bronchial or a pulmonary vessel, since it is popularly believed that if the hæmorrhage is bronchial it does not mean tuberculosis. Considerable discussion has been had on this point, but it now seems generally held that bleeding in pulmonary tuberculosis may come from either set of vessels. Thus, slight streaking of the sputa with blood may arise from extravasation, from congested bronchial or pulmonary capillaries, while it is possible for an ulcerative process to lay open either a pulmonary or bronchial artery.

In the one case, i. e., when inflammatory hyperæmia about a tuberculous focus is the cause of the bleeding (the explanation assigned by some to account for the hæmoptysis), the blood may ooze from either bronchial or pulmonary capillaries, but when the hæmorrhage amounts to several ounces and especially when frequently repeated, the source of the hæmorrhage is probably a vessel belonging to the pulmonary system, the coats of which are laid open.

Undoubtedly profuse bleeding comes in the majority of instances from a pulmonary artery or capillary, and yet there have been some observations proving that Niemeyer was not wholly wrong in attributing such hæmorrhage to the bronchial system. Sticker cites Langerhans as having quite in isolated cases, discovered the source of fatal hæmorrhage to be an of a bronchial artery. Sticker refers also to the observations of eminent Birch-Hirschfeld who believed that the earliest development cle is in the coats of middle-sized bronchial vessels close to their departure from the parent stem. He recorded one case in which th rhage came from a dilated bronchial vein that had been laid open in the tissues underlying a tuberculous ulcer in a bronchial wa instances are exceptional, however, and we may cling to the notion blood usually comes from the pulmonary system and, when prof an artery rather than a capillary.

The precise manner in which *early hæmoptysis* is produced ha been fully established. By many it is supposed due to inflammato æmia in the parts immediately contiguous to the tuberculous zon that appears to find support in the failure by pathologists always t the vessel from which the blood has escaped. It is more likely, that the hæmorrhage results from the growth of the tubercle w wall of a minute pulmonary capillary.

Cornet is of the opinion that in consequence of the breaking d tubercle a bacillus is carried into the perivascular lymph stream to s where, becoming arrested, it penetrates a capillary wall. Here it to a tubercle which impairs the integrity of the vascular coats so t heightened or perchance normal blood-pressure these rupture with hæmorrhage and hæmoptysis.

Others assume that the formation of the tubercle in the wa capillary causes a narrowing of its lumen, and as the vessel is a one without anastomoses such an encroachment upon its caliber oc localized increase of blood-pressure. In consequence of this abnor mentation of intravascular pressure a rupture takes place with a hæmorrhage that is at length only checked by the development of t within the affected capillary.

Whatever be the exact mode of production in these incipient condition, as will be seen later on, is quite different from that w rise to hæmoptysis in the later stages of the disease. To my mind monary hæmorrhage which attends and in some cases seems to tuberculosis is better accounted for by the development of the istic nodule in the wall of the vessel than by merely an active or i tory congestion, since the hæmoptysis is apt to be more profuse th seem likely in diapedesis from simple hyperæmia.

However hæmoptysis may be explained in incipient and early tu the mode of production of hæmorrhage in the stage of softening an tion seems to be well established. In *acute phthisis*, the destructi leads to rapid and often extensive cavity formation. Vessels of co size are laid bare and, their coats being weakened or even invaded cle, either rupture at such a point or are actually eaten throug same ulcerating process which destroys the pulmonary parenchyma. an event tremendous and not unfrequently fatal hæmorrhage resu this occurrence that is so dreaded in acute cases, and I have in mind more than one instance of acute tuberculous broncho-pneumonia which has so terminated.

In *chronic pulmonary tuberculosis* with cavity, the caseation proceeds slowly and time is usually given for the vessels to become obliterated and to atrophy before rupture of their walls can take place. Now and then, however, an artery of considerable lumen is left exposed in the wall of the vomica or actually traverses its interior unsupported by parenchyma (Fig. 17). When such is the case the vascular coat undergoes dilatation at some point in its course, forming a small pulmonary aneurysm, while the formation of the aneurysm is favored by the presence of tubercle in its wall. Thus is fur-

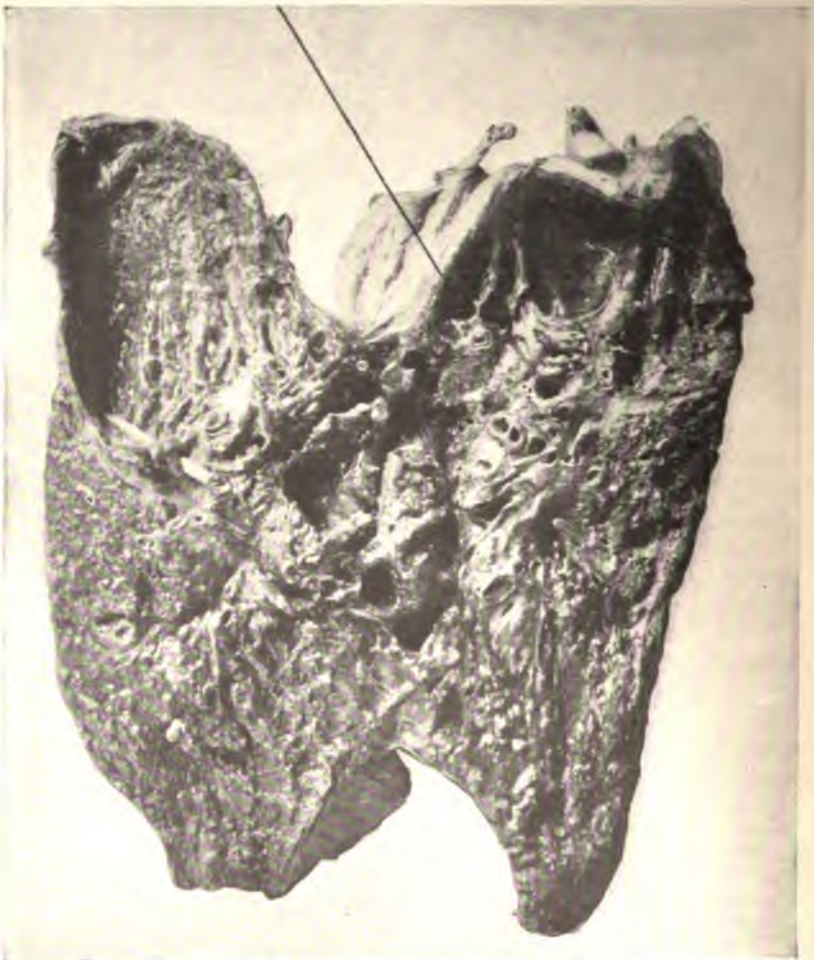

FIG. 17.—Lung with tuberculous cavity. Line marks thrombosed vessel.

nished a favorable spot for rupture whenever blood-pressure reach
a degree.

The entire volume of blood in the body has to pass through
nary system which, under normal conditions, possesses a smaller tot
than does the aortic system. Blood-pressure within the pulmon
is always relatively high, and when, on account of the tuberculo
the total vascular area in the lungs has become appreciably redu
pressure in the pulmonary arteries is abnormally high. Under
tions a pulmonary aneurysm can scarcely escape rupture, and
influence, as cough, physical exertion, constipation, etc., still fu
ments blood-pressure, the artery is very likely to yield, and h
results.

The opening of the vessel-wall thus occasioned is closed by a fl
which when hæmorrhage has lowered vascular tension sufficiently
into place and is fixed by a clot. This state of things makes it
hæmoptysis in the stage of cavity is often profuse, generally recu
seldom proves the cause of death.

(*c*) *Effect of Hæmoptysis on the Prognosis of the Underlyin*
losis.—This is to be considered from two standpoints, namely
amount of blood lost and the danger immediately following, an
more remote effects on the lungs themselves and thereby the dan

The amount of blood lost from pulmonary hæmorrhage va
wide limits depending upon the stage of the lung affection and t
cause. Initial hæmoptysis is seldom profuse. There may be a me
of the sputa with red, but usually the quantity of blood is apprec
a mouthful or two to several ounces. Therefore it falls into
category of "small hæmoptysis," i. e., up to 100 c.c. or three
very rarely reaches 500 c.c. or a pint, which this observer regards a

Although early hæmorrhages are as a rule not large they
peated at intervals of a day or two for a week or more and in th
amount to considerable. In such instances it is likely that the h
is due to something more than mere hyperæmia, in reality to em
sion of a capillary by tubercle or to loss of continuity in its
the same cause.

Repeated losses of blood in this manner naturally alarm p
friends and even create apprehension in the mind of the practition
theless, they rarely bring about the death of the patient by a
exhaustion or by asphyxia. They may, however, induce atelectasi
more areas in consequence of the aspiration of blood into depe
and such zones of collapse may favor the development of broncho-

I have recently seen in Elgin, Ill., in consultation with Dr. O.
a young married lady who had experienced four hæmoptyses of se
each in less than a week. The case was incipient, to judge fr
and signs at left apex, since there were no râles of softening. At
behind were fine and coarse bubbling râles which at the left were
ous and surrounded a dull patch over which breath-sounds were
audible. This was considered an area of pulmonary collapse. Fr

rhages were reported as taking place daily for the next five days, and this area of dullness was said to have increased in extent. At present writing it is too soon to report the outcome of this case, but as the temperature was said to be rising somewhat I fear a broncho-pneumonia will result and render most grave what otherwise might prove a relatively good prognosis.

Most instances of early hæmoptysis are not so obstinate and serious, and there is a prevalent opinion that cases of tuberculosis beginning in this manner do well. Certain it is that hæmoptysis has saved life—by which I mean of course that the expectoration of blood by serving to call attention to the pulmonary affection or by bringing a frivolous patient to a realizing sense of his danger has led to the adoption of measures which have saved his life by arresting the pulmonary tuberculosis. I have seen the truth of this statement many times.

Although repetitions of blood-spitting are the rule, still an initial or early hæmoptysis may occur but once. In my experience this is exceptional. I recall an instance in point seen not long ago. The patient was a woman of thirty with cough of several months' duration, scanty muco-purulent expectoration containing a few bacilli, moderate dullness and bronchial breathing in the left upper lobe, who declared she had had but a single hæmorrhage seven months before at the beginning of her illness.

When the local process has passed from its incipiency into the stage of softening, but may still be considered in an early stage, the amount of blood lost at a single time or, more commonly, at repeated bleedings may be truly alarming and may threaten life directly. A Chicago physician consulted me a few years ago because of cough, expectoration and loss of weight, all of the symptoms slight yet enough to create apprehension. There was unmistakable wasting of the shoulder muscles, and the right upper lobe showed dullness. Bronchial respiration was accompanied by copious fine râles after cough. Temperature was about 100° F. The process was still in the early stage. A few days subsequently just as he was preparing to seek another climate he had a considerable hæmorrhage. Not long thereafter he reached Saranac Lake, and as I have since been informed by Dr. E. L. Trudeau was seized with a second really profuse hæmorrhage soon after his arrival. In this case I do not believe the blood came from a cavity, but was probably due to rupture of some vessel whose wall had given way in consequence of its invasion by tubercle.

In *acute pulmonary tuberculosis* with rapidly progressing excavation, and in *chronic phthisis* with old cavities the results of pulmonary hæmorrhage may be very disastrous. That such should be the case is readily comprehensible when one considers that the source of the blood may be an artery of considerable caliber. Thus I know of an instance in which a consumptive, while taking his morning stroll, was seized with a tremendous hæmorrhage, fell to the ground and expired almost before his friends could reach his side. A young man returning from the country was obliged to ride through a long tunnel just before his train entered Hoboken. The gas in the tunnel as very irritating and probably excited severe cough, for just as he was emerging he was seized with a hæmorrhage. His friends carri the depot a few minutes thereafter, but he died almost as soo laid on a bench. A female patient of mine, while at dinner wit gave a slight cough and raised her napkin to her mouth just intercept the red stream that poured forth. She was unable word, reeled in her chair, was caught by her mother and died a she could be laid on the floor. In all such cases death is pro asphyxia and it is this possibility that gives so grave an aspect when hæmorrhage takes place in acute and chronic pulmonary In still other instances hæmoptysis may be excessive (one or and experienced a number of times during a lapse of months o yet no harm appear to follow. In fact it actually seems as i cases were benefited rather than injured by the loss of blood.

The more remote effects on the lungs, resulting from hæm already been incidentally referred to. Old writers and notably Niemeyer believed that pulmonary hæmorrhage might induce by the aspiration and inspissation of the blood within the alve opinion they mistook effect for cause. Hæmoptysis is not the c monary tuberculosis but is the effect, as we have seen. Yet it is q for a hæmorrhage to be a means of spreading tuberculosis throu or of carrying disease into distant parts before healthy.

This may be accomplished by the blood serving as a carrier i. e., of either tubercle bacilli or other organisms as streptococci cocci. The investigations of Perl and Lipmann have demonstrate effused into trachea and bronchi may be absorbed without setti tion, and probably such is the fact in many cases. There is liability of the aspiration of blood into terminal bronchioles in of the prolonged rest in bed insisted upon by the physician administration of sedatives to keep down cough.

Under such circumstances collapse of entire lobules may r the necessities of the case require prolonged physical inaction and of cough, there is grave danger of broncho-pneumonia. As wil the chapters on Atelectasis and on Acute Broncho-Pneumonia collapse predisposes to this form of pneumonia by favoring th germs that may be carried thither or already exist in bronchial

Consequently, bacteria conveyed by the blood into the base o bleeding or the opposite lung may take advantage of the atelectas by the aspired blood and set up a pneumonic process that may patient in a short time. In every instance of profuse and repe tysis this possibility must be borne in mind in forecasting the any given case. However, such is probably far less often the ca than is asphyxia, and it is also likely that many an instance pulmonary collapse has, in the light of the heightened temperatu hæmorrhage, been mistaken for broncho-pneumonia.

To sum up, therefore, I would say that the danger in hæm pulmonary tuberculosis lies in the amount of hæmorrhage at t in the number and frequency of its recurrences. Hence it is mo in the stage of softening and excavation although it is less liable to occur in this phase of pulmonary disease.

(2) **Bronchitis.**—Ordinary bronchitis only exceptionally gives rise to hæmoptysis and then in the chronic rather than the acute form. Now and then the cough attending an acute bronchitis may be so violent and protracted as to occasion extravasation of an insignificant quantity of blood from the mucosa when the scanty viscid sputum may display a scarcely perceptible streaking with red and may taste a little salty. This, however, is exceptional, and should an apparently simple cold on the chest be attended by the expectoration of a tablespoonful or two of blood, it should be regarded as suggestive of a tuberculous foundation for the bronchitis and lead to careful observation with this possibility in mind.

In *chronic bronchial catarrh* a tinging of the sputa with blood is somewhat more common, but even here is not usual. The congested and swollen mucous membrane may, however, permit extravasation of a small amount of blood or even the rupture of a distended capillary under the strain of severe cough. A more profuse hæmorrhage is suspicious of ulceration or of dilatation of the air-tube. In the affection known as fibrinous or croupous bronchitis, hæmoptysis even to the extent of being profuse, not seldom attends or precedes the expulsion of the cast.

(3) **Bronchiectasis.**—That this may be a cause of pulmonary hæmorrhage has been known to the profession ever since Laennec's report of two cases in which such a connection existed. Although not the rule in this disease hæmoptysis is yet relatively frequent as shown by Balzer's and Barth's statistics given by Sticker. The former observed hæmoptysis in 16 cases out of 39 of bronchiectasis and the latter in 9 out of 43 cases.

In amount the hæmoptysis is generally small, but it may be profuse and very obstinate. In 7 of Barth's 9 cases it was the cause of death. The source of the hæmorrhage may be the highly congested capillaries of the bronchial mucosa which in some zones may be so extensively dilated as to look like veritable angiomata, or very exceptionally the blood may come from a bronchial vessel of considerable size that has been laid open by rupture or by the ulcerative process so often seen in this affection. In a few instances tuberculosis is associated with the bronchiectasis and may be the cause of the hæmoptysis.

(4) **Pulmonary cirrhosis,** or chronic interstitial pneumonia, is another disease in which hæmoptysis is a not very infrequent occurrence. It may be trifling or profuse, but according to Wilson Fox was moderate in amount in 16 out of the 39 cases of chronic pneumonia in which he observed hæmorrhage. The source of the blood is in a dilated bronchus which has undergone ulceration or in a vomica of tuberculous origin, since, in a considerable proportion of cases of pulmonary fibrosis, tuberculosis is the underlying condition.

(5) **Acute pneumonia** may also, though very exceptionally, display profuse hæmoptysis. In a strict sense, the rusty and the prune-juice sputum of this complaint may be considered as the spitting of blood, but the symptom now referred to is the escape from the mouth of such an amount of

blood as to constitute a veritable hæmorrhage. This occurs most frequently at the onset of the affection, but in very rare instances the hæmorrhage may appear at a somewhat later period. Stricker is said to have recorded it but seven times in the course of 16,711 pneumonias among the German soldiery.

By some authors such profuse hæmoptysis is believed favored by the presence of tubercle, by chronic alcoholism, chronic lead poisoning and malaria. In a few instances this so-called *hæmorrhagic pneumonia* has been characterized by extravasation of blood under the skin in various parts of the body.

Vividly impressed on my memory is the case of a young man in whom profuse hæmoptysis attended the commencement of his fatal pneumonia. He had been treated under my direction with subcutaneous injections of Koch's Tuberculin-R for a circumscribed early tuberculosis of the left apex. After the treatments had been given for a number of weeks with such decided benefit that the local findings had almost disappeared and he thought himself well, he was given diagnostic injections of old tuberculin to determine whether immunity had been established or not. The first two doses were not followed by reaction, but after the third of 0.010 mg. he presented high fever and felt so ill that he took to his bed.

The next day I was hastily summoned to his bedside and learned that he had suffered a severe hæmorrhage a few hours earlier. The right upper lobe was markedly dull throughout and presented bronchial breathing where but the day previous signs of disease had been very vague. Death supervened a few days subsequently. This case may also be taken to prove the occasional occurrence of pulmonary hæmorrhage after the injection of tuberculin, as pointed out by Sticker.

(6) **Pulmonary Abscess and Gangrene.**—It is not uncommon for patients suffering from these affections to display slight hæmoptyses in consequence of the violence of their cough. It is a totally different matter, however, when destruction of lung leads to ulceration of a vessel of considerable size lying in the necrotic area. Under such circumstances, enormous and even fatal hæmorrhages may be the result. Not only have such copious hæmoptyses been observed in cases of post-pneumonic abscess and gangrene, and in such as result from embolism, but they sometimes take place from septic infarcts in pyæmia and even in the course of carbuncles (Sticker) and in diabetes, when it leads to the breaking down of a lung either with or without tuberculosis. Fox states that in some cases of gangrene hæmoptysis may be "the first overt symptom which may be repeated, and sometimes for days, before the sputa become fœtid." Such is especially likely to be the case, he adds, in the gangrene "commencing in bronchiectasis and in some cases of pneumonic origin."

(7) **Cancer of the lung** is said by Walshe to give rise to hæmoptysis in 72 per cent of cases, that is, nearly as frequently as in pulmonary tuberculosis. The sputa may be sanguineous, i. e., merely streaked or mixed with blood, but, according to the same author, amounts exceeding one ounce are nearly twice as common (in the ratio of 70 to 40) as in phthisis. Fox states that pulmonary hæmorrhage in cancer of the lung sometimes is so abundant as

to prove the immediate cause of death. In most cases, however, staining of the sputa with blood is more common than hæmorrhage and, according to Lenhartz, fatal hæmoptysis is extremely rare.

(8) **Pulmonary infarcts** are also characterized by the spitting of blood which is bright red and comes up as pure blood unmixed with mucus. It is observed chiefly in cases of chronic heart disease. Minute infarcts of a septic nature may or may not be attended by the expectoration of blood.

(9) **Pulmonary Syphilis.**—Active destruction of the lung in this disease is of great rarity, apart from tuberculosis, and hence it is scarcely to be reckoned with as a cause of pulmonary hæmorrhage. Nevertheless, the possibility of profuse and even fatal hæmoptysis in the course of lung syphilis in consequence of excavation and erosion of a pulmonary artery is proven by the case reported by Remsen from the Pathological Laboratory of Johns Hopkins Hospital, 1903.

(10) **Chronic Heart Disease.**—Staining of the sputa with blood is very common in cases of pulmonary stasis resulting from chronic cardiac affections, particularly in mitral stenosis. It is due, probably, to extravasation from the congested bronchial mucosa. I have known such slight hæmoptysis on account of the associated cough to create a suspicion of incipient tuberculosis, and hence, in all cases of hæmoptysis, careful examination of the heart as well as of the lungs should be made.

When, in a given instance of cardiac disease, hæmoptysis becomes more profuse it is probably the result of pulmonary infarcts or of such an infiltration of the parenchyma with blood and consequent laceration of the tissues as to merit the term pulmonary apoplexy. The emboli causing the infarcts in such cases arise from stagnation of blood within the overdistended right auricle and denote grave cardiac asthenia from which the patient is scarcely likely to recover. I have under observation a young man with mitral regurgitation, who several times has experienced small hæmoptyses. They are not accompanied or followed by fever, and repeated examinations of the lungs show no signs of tuberculosis. There are dilated venules in the throat and these are suspected of being the source of the hæmorrhage.

(11) **Aortic aneurysm** occasionally is a cause of hæmoptysis which, in its amount, depends upon the condition responsible for its occurrence. The sac may rupture into a bronchus when the resulting bleeding is so profuse as to prove speedily fatal. The expectoration of small quantities of blood which may be frequently repeated over a period of many weeks is owing to congestion of the bronchial mucosa, to gradual destruction of lung from pressure, to oozing from tracheal granulations or to weeping of the aneurysm itself through the laminæ of coagula which, in some cases, at length come to form the wall of the sac.

(12) **Arthritic Diathesis.**—Writers generally do not appear to discuss the occasional occurrence of pulmonary hæmorrhage in that type of individuals described by Lancereaux and other French writers as arthritic. At least I fail to find references to it in most of the works at my disposal. Osler mentions it and Sticker, to whose exhaustive article on "Lungenblutungen" in Nothnagel's System I have so frequently referred, deems it worthy of

consideration in spite of the skepticism of his countrymen. I have not myself observed such an instance of hæmoptysis, and yet I remember being consulted some years ago by an Englishman of the arthritic type who declared he had experienced a profuse pulmonary hæmorrhage which was attributed by his physician in England to gout.

This form of hæmoptysis was recognized by Sir Andrew Clark who in 1889 contributed a paper to the *British Medical Journal* under the title, "Remarks on the Nontubercular and Noncardiac Hæmoptyses of Elderly Persons."

The hæmoptysis is observed chiefly in elderly men who present signs of chronic bronchitis and emphysema together with gouty arthritis. The source of the bleeding appears to be in the terminal pulmonary arteries which, in consequence of hyaline degeneration of the intima and media, have become occluded, with resulting stasis in the neighboring venules and capillaries with areas of atrophic emphysema.

Such were the findings in a man of about fifty years who, admitted to the hospital with history and signs of subacute bronchitis with emphysema, at length began to have frequent small hæmoptyses at short intervals. Fever was slight, heart and arteries were negative, and yet after spitting blood for a week he succumbed. The autopsy disclosed no tubercles.

(13) **Certain depraved states of the blood,** as purpura hæmorrhagica, scorbutus, progressive pernicious anæmia in which hæmorrhages are frequent in various parts of the body, may also lead to loss of blood from the bronchial mucous membrane and hæmoptysis.

In this connection it may also be stated that pulmonary hæmorrhage occasionally occurs in some of the acute infections, e. g., typhoid fever. Enteric fever displays a special tendency to pulmonary complications, viz., bronchitis, pneumonia and pleurisy, which in the presence of such profound disturbance of nutrition as is the case in this infection may readily favor hæmorrhage from the mucous surface of the air-tubes. Nevertheless, Sticker's explanation of the hæmoptysis infrequently seen in *grippe* seems to me pertinent to its occurrence in typhoid fever. He suggests that when, in influenza and other acute infections, pulmonary hæmorrhage results, not as a primary symptom of pulmonary involvement but in consequence of the traumatic action of the infectious agent on the lungs, there is some old-standing local lesion, as tuberculosis, that serves as a predisposing factor. In support of his contention he quotes Stricker's observation that of the 178 cases of hæmoptysis occurring among 3,185 soldiers suffering from influenza the majority concerned those men who were tuberculous.

(14) **Distomum Pulmonalis.**—Notwithstanding the existence of this parasite in North America, it possesses but little interest to us of the United States as a cause of hæmoptysis, although Naunyn observed an instance in a German who had lived for fifteen years in Mexico and California (Fraenkel). In Japan, Korea, Formosa and the islands of the archipelago to the south of Formosa it constitutes an extremely common and incurable cause of pulmonary hæmorrhages associated with cough and a scanty viscid sputum. The general health does not appear always to suffer and the hæmoptyses are

usually scanty. Under exertion, however, the quantity of blood thus lost may reach a pint or more. For the knowledge of this form of hæmoptysis we are indebted to Baelz whose original paper on the subject appeared in 1880 (*Zentralbl. f. d. ges. med. Wissensch.*, Nr. 39).

(15) **Vicarious Menstruation.**—From the time of Hippocrates to the present the belief has been current in the profession as well as among the laity that suppression of the menses or of a hæmorrhoidal flux is sometimes followed by epistaxis or hæmoptysis. It is also the opinion that such vicarious hæmorrhages are beneficial rather than hurtful by relieving the plethora and congestion that otherwise might occur. Personally I have always been rather skeptical on this matter, and hence am pleased to find my skepticism shared by Sticker.

Nevertheless, there are a few cases of the kind in the literature which have been recorded by physicians of such experience and repute that it would smack of arrogance to deny flatly the possibility of such an occurrence. Yet, even granting that hæmoptysis may supervene upon the cessation or diminution of menstruation, it cannot be proven that the blood-spitting was really vicarious in its nature. The occurrence of the pulmonary hæmorrhage is to be explained in some other way. When it follows catamenial suppression by fright, shock, excitement, etc., it may be due to circulatory or nervous disturbance in consequence of which the lungs become temporarily congested. Such sudden hyperæmia may readily induce rupture of a distended vein or capillary within the trachea or large bronchi, and the observer may not be able to determine the precise seat of the bleeding. Sticker believes that in many of these cases there is a tuberculous focus within the lungs.

(16) **Influences from the Side of the Nervous System.**—A great number of experiments on animals and numerous clinical observations have shown beyond cavil that pulmonary ecchymoses and even hæmorrhage may result from injury to various parts of the central nervous system, pons, spinal cord, etc. There are also cases on record in which pulmonary hæmorrhage took place in connection with insanity, epilepsy or hysteria, without the demonstrable presence of tubercle as shown by *post-mortem* examination and the result of treatment.

A notable instance of hysterical hæmoptysis is narrated by Debove of a medical student who, for a long time, had recurring hæmoptysis and on that account was held to be tuberculous. At length the discovery was made of a right-sided hemianæsthesia together with other signs of hysteria. Appropriate treatment was instituted and complete recovery of health ensued.

In this connection may be mentioned the rare appearance of blood-spitting in young, impressionable persons at or near the age of puberty, who belong to the type designated as erethismic. Such individuals are often the children of tuberculous or syphilitic parents and are distinguished by delicacy [illegible] abnormal irritability of the nervous system and a marked inclination to [illegible] congestions or fluxions. Under this category doubtless belong some [illegible] individuals who are said to be natural bleeders or have the hæmor-

May it not be that to this class belong those instances mentioned by Osler of young persons who manifest hæmoptysis yet maintain good health for many subsequent years, without developing signs of pulmonary disease? In support of this statement he cites Ware's figures according to which 62 out of 386 cases of hæmoptysis failed to show subsequent evidence of pulmonary lesions.

It would be interesting if it could be ascertained in how many of such cases the ultimate failure of tuberculosis to declare itself was due to the better care of the health taken after the hæmoptysis in consequence of the dread of the disease aroused by the bleeding. In all cases in which the hæmorrhage is attributable to nervous influence, strictly speaking, there is probably profound vaso-motor disturbance since vascular dilatation favors the occurrence of hæmorrhage, and normal capillaries will stand a pressure of 65 cm. of mercury without rupture.

Several years ago I attended a young man with a bad family history who, while walking along the street, gave a slight cough and spat up a mouthful of bright red blood. I had examined his chest the day previous and been unable to detect evidence of disease. Upon the occurrence of his blood-spitting, therefore, he at once hastened to my office when I again examined his chest without being able to discover the site of the hæmorrhage.

He was reassured and told to go about his business but to be careful not to overexert himself. A few hours subsequently he again perceived a slight irritation in his chest or throat and again expectorated blood in the same amount and of the same appearance as in the morning. He betook himself to bed in considerable alarm and summoned a physician.

I saw him the next day and was totally unable to locate any pulmonary disease. During the succeeding two weeks he expectorated some dark clots and experienced a third slight hæmorrhage immediately after some exertion that coincided in time with his being startled by a sudden severe flash of lightning and a closely following clap of thunder. After this hæmoptysis, I detected some indistinct râles in the first right interspace not far from the sternum. It seemed to me also that resonance in this region was a trifle impaired.

It was reluctantly concluded that there must be a tuberculous foundation for the symptom and the patient was sent off to Colorado. He went to Dr. W. H. Bergtold in Denver who, not being able to locate a definite lesion, administered tuberculin in full doses, but without obtaining reaction. Nevertheless the man remained away for a number of months until his health and weight became far ahead of what they had ever been.

Since that time I have repeatedly examined this man and have never been able to detect positive evidence of pulmonary disease. What now was the cause of the hæmoptyses in this case? Was there an ulcer or a dilated venule in the trachea or one of the main bronchi which under the influence of exertion emitted a small amount of blood? If so it could not be discovered. Or was there a tuberculous focus too small to give reaction to tuberculin? I recall a dentist of about forty who had hæmoptysis which could not be traced to the lungs, and in whose trachea such a network of distended capillaries could be seen as to suggest one of these vessels as the source of the blood.

Symptoms.—The occurrence of pulmonary hæmorrhage is usually sudden and without premonition. During sleep, it may be, or while making some exertion the individual gives a slight cough, perceives a salty taste in the mouth and spits out a mouthful or two of bright red blood which, as it generally contains air, is somewhat frothy. If the sputum is merely sanguinolent and not made up of pure blood, it consists of a mixture of saliva, mucus and epithelial cells, with the cellular elements of blood and micro-organisms.

In profuse hæmoptysis the blood gushes or pours from the mouth in a bright stream greatly to the terror of the friends as well as of the sufferer. Such copious hæmorrhages may prove fatal by flooding the lungs and interfering with respiration. If the blood pours into a large cavity it may cause syncope, either temporary or fatal, without revealing the enormous amount of the blood that is lost. Commonly, however, the hæmorrhage recurs one or more times at varying intervals for days or weeks until the invalid becomes almost exsanguinated or actually dies of exhaustion. I knew a case in which, during two or three weeks, at least four such enormous hæmorrhages took place, the patient dying suddenly in the last one. Laennec and J. Frank are said to have observed hæmorrhages of ten to twelve pounds in twenty-four hours, and even thirty pounds in two weeks.

Such enormous hæmorrhages rarely take place in any other affection than advanced or rapidly progressive phthisis, and owe their origin to rupture or erosion of a pulmonary artery. The escape of blood from an aortic aneurysm is so overwhelming as to leave no possibility for repetitions as in tuberculosis. In all cases of hæmoptysis, however, the amount of blood expectorated is determined by the condition responsible for its occurrence.

In cases of pulmonary stasis from cardiac disease and in bronchitis, etc., the quantity of blood coughed out is generally scanty, although in pulmonary embolism or apoplexy the hæmorrhage may sometimes prove considerable. In pulmonary tuberculosis the quantity of blood spat up varies greatly, from a mere staining of the sputa with red to the profuse amounts already mentioned.

There may be but a single hæmoptysis, but as a rule the bleeding is repeated from time to time or, continuing, gives rise to bloody sputa during days and even weeks. For the most part the first hæmoptysis is succeeded by the coughing up of dark-colored clots, sometimes at intervals during the day or on successive mornings until at length the bleeding ceases altogether. The character of the expectorate usually changes, becoming darker colored and clotted and gradually diminishing in amount day by day.

From the foregoing it is apparent that the greatest possible differences are presented by cases as regards the *quantity* of the hæmoptysis. It may vary from a mere staining or streaking of the sputa with red to amounts that are really enormous. Patients are very apt to exaggerate the quantity not always from a desire to magnify but because the impression created is such that the amount appears larger than it really is. For the sake of possessing some criterion by which to classify pulmonary hæmorrhages as small, moderate or excessive and thus be able intelligently to meet inquiries on this score, we may take the figures given by Stricker.

He considered as small amounts less than 100 c.c. (3 ounces), as moderate from 100 to 500 c.c. (from 3 to 16 ounces) and as large, all amounts over 500 c.c. For my part, I should be inclined to call a pint (500) large and quantities in excess of this figure as excessive.

Auscultation soon after a copious hæmorrhage from the lungs may disclose fine bubbling râles over a circumscribed region usually below one or the other clavicle, and if a pulmonary embolism has caused the hæmorrhage it may in some cases be possible to detect, by careful percussion, an area of dullness near the base of the affected lung. The caution should be expressed, however, that as a general rule it is not safe to perform percussion on the chest of one who has just had hæmoptysis.

No matter what the condition underlying the hæmorrhage, the individual is usually thrown into a state of much excitement and, it may be, of great apprehension. The pulse is accelerated and often bounding, and the skin is warm and moist unless perchance the quantity of blood lost is excessive when the patient may present the cold clammy surface and pinched anxious countenance of collapse. In this condition the pulse is small and weak, the respirations shallow and the voice faint and husky.

If the individual rallies from the depression incident to profuse hæmoptysis, the reaction, especially in tuberculous patients, is likely to be characterized by *fever.* This may be moderate and persist for a few days to a week or it may reach 103° or even 104° F. in cases in which an aspiration pneumonia ensues.

Hæmoptysis from other than pulmonary diseases, i. e., from cardiac or vascular disease, vicarious menstruation and nervous influences, is not succeeded by a febrile temperature, and so far as it affects the general state occasions nothing more than a psychical disturbance depending in degree upon the person's impressionability. It is in pulmonary affections, and of these chiefly in acute or advanced phthisis with cavity formation, that pulmonary hæmorrhages produce effects so serious as to become in themselves a source of danger, namely, *extreme anæmia* and profound exhaustion.

Diagnosis.—In a strict sense this does not concern the fact of hæmoptysis for this is established by the declaration of the person himself or of the bystanders, but has to do with the source and cause of the bleeding. In many instances this is a matter of no great difficulty since the individual may be known to have consumption or some other affection of lungs or heart capable of occasioning hæmoptysis; but every now and then cases are encountered which tax the physician's diagnostic acumen or must remain doubtful until time clears up the nature of the hæmorrhage.

In all instances where there is doubt whether or not the lungs are the source of the bleeding careful inspection must be made of the nose, throat and mouth for the detection, if possible, of some bleeding point, as a ruptured venule at the root of the tongue or in the post-nasal space. The gums should also be closely scrutinized since persons have been alarmed by the discovery, on the pillow, of blood which oozed from the unhealthy gums during sleep, or by suddenly perceiving a salty taste in the mouth and thereupon expectorating bloody saliva.

In all cases, therefore, of scanty hæmoptysis minute inquiry should be made concerning the manner in which the blood was ejected as well as concerning its appearance. If it comes from the upper respiratory tract or the mouth, its appearance in the mouth may not be attended by cough, and it is not likely to be mixed with bubbles of air; whereas hæmoptysis in consequence of disease below the glottis is quite sure to be preceded by or accompanied with cough either with or without tickling or other sensation in the upper part of the chest. Moreover, as is usually stated by authors, the blood is of a bright red hue and when scanty is very likely to be frothy from its containing air.

Although cough may at times be occasioned by the escape of blood from a small vessel on the wall of the pharynx, still this symptom in conjunction with hæmoptysis strongly suggests the lungs as the seat of the hæmorrhage. Notwithstanding this fact however the physician should make it an invariable rule to examine the larynx and trachea as well as the parts above in every case of hæmoptysis which does not furnish *prima facie* evidence of having come from within the depth of the chest.

Even a copious pulmonary hæmorrhage may be ushered in by cough, but it need not be attended by coughing since the freedom with which the stream flows into and out of the mouth does not permit such an act.

In cases of scanty as well as profuse hæmoptysis, but especially in the latter, much examination of the chest is not advisable and hence an attempt to arrive at diagnosis from the anamnesis should be made in every instance. If it is impossible to reach a satisfactory conclusion, then, in cases not evincing much gravity, auscultation of the chest may be practiced. Percussion is not necessary, as a rule, and would better be left for a subsequent occasion. When the hæmorrhage has been excessive it is for the most part better not even to resort to auscultation, lest the psychical disturbance it sometimes occasions may bring on a repetition of the bleeding. This caution is certainly wise in cases of undoubted pulmonary hæmorrhage from tuberculosis.

In those instances in which the hæmorrhage may be thought due to some other than tuberculous disease, a cautious examination of the chest, in particular of the heart, is admissible for the purpose of deciding on the plan of treatment. It would be out of place here to detail the signs by which the various conditions capable of causing hæmoptysis may be recognized, but it may be stated that the recognition of cardiac or pulmonary disease furnishes presumptive evidence in favor of the lungs as the source of the hæmoptysis.

Hæmatemesis is a possibility that should always be borne in mind in every case of bleeding from the mouth. As a rule, the patient is able to state definitely whether or not the blood came with the act of vomiting and thus to aid materially in settling the diagnosis. When doubt still remains, or when there is a possibility of an hysterical patient having intentionally swallowed blood for the purpose of deception, or when coughing and vomiting both are present, then the following differential points may be borne in mind.

(1) Vomited blood is usually of a dark brown color instead of bright red and frothy, and may look not unlike coffee grounds. (2) It is acid in reaction while that of hæmoptysis is alkaline. (3) Hæmatemesis is apt to be succeeded by blackish stools looking not unlike tar. (4) The microscope may detect particles of food mixed with the vomitus, while in pulmonary hæmorrhage there may be muco-pus and perhaps bacilli contained in the clots.

As a rule the differential diagnosis is not very difficult, but now and then cases arise in which the determination of the nature of the malady is most puzzling or even impossible. Thus the rupture of dilated œsophageal veins secondary to atrophic cirrhosis of the liver may cause hæmorrhage which, occasioning cough and possessing a red appearance, may closely simulate hæmoptysis. It is likely, however, that blackish blood will be vomited before the hæmorrhage ceases wholly, or that the alvine discharges will present evidences of blood having passed down the intestinal tract.

In illustration of the difficulty occasionally underlying the discovery of the source of hæmorrhage because of similarity in the clinical picture possessed by wholly different diseases, Sticker cites the case reported by Honigmann which, condensed, is as follows: A railroad engineer, aged fifty, who had some years previously suffered with malaria, and for the last year with ill-defined stomach trouble, was seized in the night with cough and hæmoptysis. The blood was bright red and frothy and produced the sensation of coming from directly beneath the sternum which supposition seemed confirmed by the detection of a small area of impaired resonance at the right apex. Moreover, in the next few days small clots were several times expelled which were forked and looked as if they might have emanated from the bifurcation of the trachea.

He repeatedly had chills with subsequent rise of temperature to 104° F., and with palpable enlargement of the spleen. He also had a second hæmorrhage which was frothy and accompanied by coughing. In a word, the case seemed to be one of pulmonary hæmorrhage.

The autopsy however disclosed, in addition to a small peribronchial focus in the right upper lobe, beneath the clavicle with a slight degree of emphysema but without evidence of blood in the air-passages, an abscess cavity situated underneath the diaphragm and back of the cardiac orifice of the stomach. This cavity which was filled with decomposing blood and pus communicated with a similar one in the upper posterior part of the spleen. It also opened into the œsophagus by means of a minute perforation that appeared to have been produced by an ulcerative process.

The actual source of the hæmorrhage does not appear to have been settled even by the necropsy. The cough and appearance of the blood certainly
m to indicate the lungs as the seat of the hæmorrhage, and yet it was
for the blood to have come from the abscess cavity.

–This can scarcely be considered independently of the patho-
nderlying the hæmoptysis, since upon this must depend
d remote seriousness of the hæmorrhage. Nevertheless,

there are two points that must always be considered in attempting to make a prognosis in any given case of pulmonary hæmorrhage.

(1) What is the gravity attaching to the hæmoptysis *per se*? Except in cases of aortic aneurysm and such diseases as lead to destructive changes in the lungs, i. e., pulmonary tuberculosis, cirrhosis, bronchiectasis, etc., hæmoptysis is seldom if ever so profuse as to weaken the patient or endanger life. In bronchitis and cardiac disease the coughing up of blood now and then may be said to be without danger, and hence such gravity as attaches to it comes from the consideration that it may indicate stasis in the pulmonary and bronchial vessels.

In thoracic aneurysm the expectoration of even a small quantity of blood should always be regarded seriously as being the possible precursor of a fatal hæmorrhage at any time. In pulmonary tuberculosis the stage of the disease may be said to determine the gravity of even a small hæmoptysis. In its incipient or first stage destructive lesions are not so likely as after softening and ulceration have set in, so that pulmonary hæmorrhage rarely reaches alarming proportions. Yet, inasmuch as we are not able to say just what is the condition directly responsible for the hæmoptysis, it should never be lightly regarded, no matter how scanty.

The same may be stated also concerning hæmoptysis in cases of bronchiectasis and lung syphilis, abscess and gangrene. In acute pneumonia, on the other hand, hæmorrhage is rarely if ever dangerous.

Profuse pulmonary hæmorrhages always carry with them the possibility of death from suffocation, exhaustion or subsequent broncho-pneumonia. This is particularly true of pulmonary tuberculosis since the blood may come from a vessel of such caliber as to make its closure by a clot practically impossible. Nevertheless, death during or directly after a pulmonary hæmorrhage is relatively uncommon when the frequency of large hæmoptyses in tuberculosis is considered.

(2) What is the prospect of a recurrence? In the vast majority of all cases of hæmoptysis from whatever cause, a repetition of the blood-spitting is likely. The number and frequency of recurrences, however, are apt to be determined by the pathological state. In phthisis and other diseases which are likely to occasion rupture or erosion of a good sized vessel the hæmorrhage is very likely to be repeated and may defy all efforts toward its complete suppression. It is the rare exception when but a single hæmoptysis is experienced, and hence the patient or his friends should be told to expect more than one hæmorrhage. Such recurrences may be at short intervals, or months and even years may intervene.

Treatment.—Two main indications are present for the management of cases of hæmoptysis: (1) the arrest of the bleeding, and (2) the prevention of its recurrence. Large hæmorrhages from any part of the body tend to spontaneous cessation so soon as the reduction in the volume of the blood permits contraction of the injured vascular coats and closure of the opening that allowed the bleeding. This applies to the pulmonary and bronchial vessels as well, but before nature thus checks a dangerous hæmoptysis the sufferer may become asphyxiated or sink i[illegible]e.

After spontaneous arrest of a hæmorrhage takes place nature endeavors to guard against its repetition by plugging the wound in the vessel-wall by means of a coagulum or by sealing together the edges of the opening. In the case of the pulmonary system variations in blood-pressure are so easily produced and arterial tension as a secondary result of the destruction of other portions is so high in the parts of the lung still remaining healthy, that the dislodgment of the clot or the reopening of the wound in the vessel-wall becomes an easy matter.

Again, our pharmacopœia possesses no remedies which exert a selective action upon the vessels within the lungs, as is proven by the enormous number of drugs that have been tried in a purely empirical way and which, for the most part, have been proven worthless if not actually harmful. In particular we have no medicament capable of directly constricting the pulmonary capillaries, and hence attempts to contract them must be through means directed to stimulation of the sympathetic.

Experiments have shown that pulmonary anæmia can be produced through stimulation of the upper five dorsal sympathetic nerves either by irritation of their roots or by application of the stimulus to the peripheral nerves. And what is of special significance, sensory impressions made upon the intercostal nerves exert a reflex influence upon the vaso-constrictors of the lungs.

The foregoing considerations make it clear that our therapy in cases of pulmonary hæmorrhage is limited, and that such results as may be accomplished by means of drugs are achieved indirectly. It is especially plain that even more is to be achieved by hygiene and other measures than by drug-therapy.

(1) **The Arrest of the Hæmorrhage.**—Desirable and even necessary as this may be in all cases, yet in scanty hæmoptysis from whatever cause, even in pulmonary tuberculosis, this is not a matter of moment when it is considered that the loss of blood is not in itself dangerous. This indication may be regarded as subordinate for the time to that of quieting the patient's fears and calming his excitement, especially as measures to this end also tend to check the cause of his alarm.

The first duty of the physician therefore is to assure the individual that he is in no immediate danger since the amount of blood lost is trifling. Even when the hæmorrhage is extreme the patient is to be reassured in the same manner since, as a matter of fact, death during or directly following the hæmoptysis is not usual. Should the patient remain excited and restless, or should his agitation be so pronounced as to be likely to maintain or intensify the bleeding, then positive therapeutic measures may be advisable.

When a drug is necessary the promptest and most efficient is *morphine* $\frac{1}{8}$ to $\frac{1}{4}$ grain injected beneath the skin. This remedy not only exerts a calmative effect speedily, but it tends to arrest the bleeding through its influence on the vascular system; at the same time it allays the cough which, being present in most cases, maintains the bleeding.

Morphine or opium administered *per os* is both slower of action and more likely to disturb the stomach. If for any reason a hypodermic of morphine is not admissible, then heroin $\frac{1}{6}$, codeine $\frac{1}{2}$, or dionine $\frac{1}{4}$, may be given by mouth.

The patient must also be enjoined not to talk lest he excite cough, but especially lest he promote fresh hæmorrhage in consequence of the heightened intra-alveolar pressure incident to the act of speaking. Words are uttered only when expiratory effort is exerted, and under such circumstances blood-pressure within the pulmonary capillaries is augmented. This certainly favors bleeding from the vessels within the lungs.

The individual should of course be commanded to remain at *complete physical rest,* preferably in a semirecumbent posture on couch or bed. If the hæmoptysis occurs at a distance from his home he should be there kept at rest until the hæmoptysis has ceased, and only then should be permitted to go to his home in a carriage. Such precaution may seem extreme, but in cases of tuberculosis it is impossible to predict that a trifling hæmorrhage at first may not under exertion be succeeded by a larger or even serious one.

The spitting of small amounts of blood does not ordinarily call for the administration of medicines popularly supposed to be of service in pulmonary hæmorrhage, since there are other means equally if not more efficient. Of these the best is said to be the application of *cold to the chest.* Its *modus operandi* appears to be by reflex stimulation of the vaso-constrictor fibers of the upper five dorsal nerves of the sympathetic system, and therefore the cold must be applied in such a manner as powerfully to impress the sensory filaments supplied to the skin of the thorax through the intercostals.

A small napkin or large washcloth folded so as to form a square of four to six inches is wrung out in ice water and then laid on the bare surface of the front of the chest. This is covered by another dry cloth and allowed to remain *in situ* for half a minute after which it is replaced by a second wet one also permitted to remain but thirty seconds. As the local sensitiveness to cold is lost after the lapse of a minute, the compress is then removed and applied in another situation. In this manner several areas may be subjected to the stimulating effect of the cold, care always being exercised not to suffer the applications to remain so long in any region as to have their effect succeeded by the capillary paresis occasioned by too protracted contact with this means of refrigeration.

There is nothing new in this procedure since it has been employed for many years by hydrotherapists of Europe. Thus Fernet, who claims to have used it in the manner described for the past fifteen years, declares he has repeatedly witnessed its beneficial effects not only in cases of pulmonary congestions but likewise in hæmoptysis, even when severe. The only caution to be observed is in cases showing profuse perspiration, and yet he does not hesitate to employ cold compresses even in such instances.

B. O. Kinnear in the *Brooklyn Med. Journ.,* Oct., 1897, highly recommends the application of a *hot-water bag* at 115° F. over the dorsal vertebræ (Fig. 18), claiming that it powerfully contracts the arterioles of the small bronchi and lungs. Greater heat than 120° may be dangerous to weak hearts. In copious pulmonary hæmorrhage when blood pours from the mouth in a stream and threatens to asphyxiate, cold compresses may be employed and will doubtless prove of service, but as their action is too slow for safety, some additional treatment is indicated.

Undoubtedly the most efficient means for the arrest of such profuse hæmoptyses is the *subcutaneous injection of $\frac{1}{50}$ and even $\frac{1}{25}$ of a grain of sulphate of atropia.* There is nothing dangerous in such doses, as I have repeatedly proven in my own experience, and Dr. S. E. Solly whose great experience entitles him to speak with authority has assured me that in these serious cases he always resorts at once to the hypodermatic administration of $\frac{1}{25}$ of atropine. Its effect is the speedy dilatation of the superficial capillaries, as shown by the redness of the skin throughout the body, and hence the remedy bleeds the patient into his own vessels, as it were.

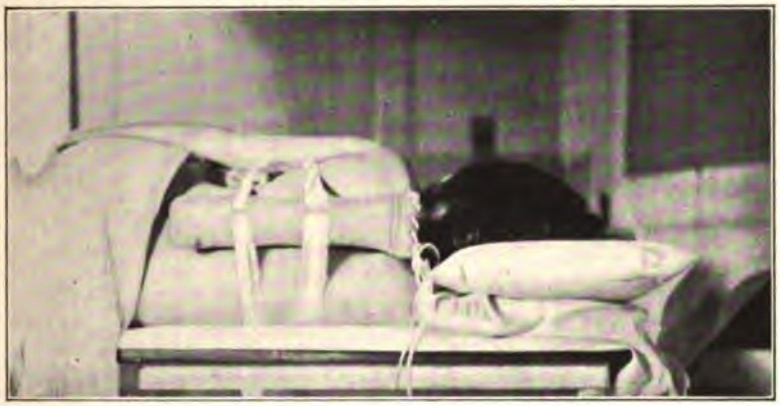

FIG. 18.—Method of application of spinal hot-water bag.

The pulse at once grows soft and full, and as the tension within the pulmonary vessels is secondarily influenced by that of the aortic system, the vaso-dilatation caused by the atropine lowers blood-pressure in the lungs. Certain it is that this medication has many times saved human life. If deemed advisable it may be injected along with $\frac{1}{4}$ of a grain of morphine, but the dose of the atropia salt should be at least $\frac{1}{50}$ of a grain.

This treatment is of course attended by all the disagreeable physiological effects, dimness of vision with mydriasis and dryness of the fauces, but these are of infinitely less moment than is the danger attaching to continuance of the free hæmorrhage.

Should the atropine in the manner just recommended not arrest the hæmorrhage it is not to be repeated during a single bleeding, but must be followed by other measures as cold to the chest in the mode described, heat to the upper dorsal region, large sinapisms to the feet and legs, ligation of the extremities and faradization of the skin of the thorax. The swallowing of table salt, a spray of a weak watery solution of subsulphate of iron and the hypodermic injection of ergot are all useless or worse than useless. Ergot in particular is injurious since by constricting the arterioles of the aortic system it raises blood-pressure within the pulmonary circuit and may actually aggravate the hæmoptysis.

Faradization of the skin of the chest is highly recommended by some clinicians but by Fernet is declared no more efficient than his method of applying cold. It acts by stimulating the intercostal nerves and thus producing reflex irritation of the vaso-constrictor fibers of the sympathetic. The caution should be exercised not to apply the current to the same spot for too long a time since it might then exert an opposite effect the same as does the too prolonged administration of cold.

Should these various methods not succeed in permanently checking hæmorrhage I know of no others likely to prove of greater service. The *subcutaneous injection of gelatine* as in the treatment of aortic aneurysm has been tried and by some been warmly recommended. Fraenkel saw hæmoptysis cease promptly in several instances but does not feel at all sure that it may not have been an illustration of *post hoc* rather than *propter hoc*. The treatment is open to several objections, such as the painfulness of the injections, the risk of tetanus poisoning and the production of high subsequent fever; so that it is to be resorted to only in cases of daily and exhausting hæmoptyses which threaten the life of the patient.

The *constriction of the diseased half of the chest* by means of adhesive plaster has also been employed and warmly recommended. The plaster may be laid on in overlapping strips or in any other manner which will restrict the respiratory movements of the affected half while leaving the other unimpeded. The bandage should be adjusted snugly and with the chest in the position of expiratory collapse. The rationale of the treatment seems to be the securing of immobility of the lung and hence lessened activity of blood-flow through the injured vessel. It is conceivable, as it seems to me, that under some circumstances blood-pressure might be raised and a hæmorrhage increased, but such an unfortunate effect is denied by advocates of this method.

The *ligation of the extremities* is an old-time plan of treatment which is said to be of use in refractory cases. The bandage must not be so tight as to extinguish the arterial pulse, but merely impede the return flow to the right heart. It is to be kept on for an hour or so and then removed slowly lest the sudden release of the blood confined in the veins flood the lung so to speak and do still more damage.

(2) **Prevention of a Recurrence.**—In many respects this is the most difficult part of one's management of hæmoptysis, especially in cases due to destructive changes in the lungs. Bloody expectoration from mitral stenosis or other forms of cardiac disease are generally amenable to measures directed to relieving stasis, and for obvious reasons these will not be considered in this work. Neither also are given the measures indicated in any of the other conditions that do not properly belong to a treatise devoted to pulmonary affections.

In the overwhelming majority of instances the recurring hæmoptysis so dreaded by physician and patient alike occurs in tuberculosis of the lungs. It is met with chiefly in chronic cases, and the pathology of the condition shows how likely is repeated bleeding in such cases. We cannot directly ligate a pulmonary or bronchial artery, and consequently we are limited to such a plan of management as will promote healing in accordance with nature's methods.

We are not in possession of remedies which increase the coagulability of the blood with such certainty as renders them absolutely reliable in such an emergency as the one now confronting us. Gelatine may be given as food and by some is said to be as efficient as its hypodermic injection. Chloride of calcium in repeated doses may also be administered internally and will probably prove as powerful in this direction as anything. This is especially advocated by W. E. Dixon. Its coagulating effect on the blood is said to be lost after twenty-four hours, and hence it is useless to continue the remedy after that length of time. Acetate of lead and digitalis (Traube's old stand-by), oil of turpentine by mouth, copaiba and the like have had their advocates but are, as a matter of fact, of no real use in preventing frequent recurrences when the blood comes from a pulmonary aneurysm or an ulcerated artery, the opening in the wall of which is too large to be securely closed by a clot.

In reality we are limited to measures calculated to constrict the lumen of the bleeding vessel and to reduce blood-flow or blood-pressure within the artery to as low a point as possible. It is so easy a matter for the wound in the vascular wall to be reopened that fresh hæmorrhages are likely to occur before the edges of the torn coats can be firmly united. On the whole, therefore, we are compelled to rely on indirect methods.

Suprarenal extract is the agent which, theoretically at least, ought to accomplish the end desired, but I have had no experience with it in this class of cases. It may be given hypodermically, by mouth, or dissolved on the tongue in considerable doses and at rather short intervals. Its effect upon aortic blood-pressure should be watched and the remedy discontinued when the latter becomes much raised, lest the tension of the pulmonary vessels be secondarily exalted. *Its hypodermic administration is not without danger, since sudden death has been known to occur.*

In all cases the *bowels must be kept relaxed* by the daily administration of an aperient water or a laxative pill. The purpose of this is to favorably affect pulmonary circulation through stimulation of the splanchnic nerves which are known to regulate blood-pressure throughout the body. At the same time there may be something in the old-time notion that laxatives exert a derivative effect on the blood-stream tending to draw it away from the lungs, since it certainly lessens stasis within the pulmonary system. The caution should be observed however to avoid strong physic for these patients, inasmuch as tuberculous subjects are very easily weakened by free catharsis.

The *use of morphine* is good treatment and is very general, but I doubt if the *rationale* of its employment is always understood. It is usually administered for the restriction of cough which, if frequent or severe, tends to bring on a return of bleeding. Such is necessary especially soon after a large hæmoptysis, but it is not the sole or chief purpose of morphine in these cases. Its main benefit comes from its ability to slow the respiration and thus favor recovery by retardation of the flow and coagulation of the blood within the bleeding vessel. Accordingly, morphine should be given in as large doses and at such intervals as may be necessary to keep the respirations down to twelve or even seven in the minute.

This action of morphine should be kept up for three or four days after a profuse pulmonary hæmorrhage. It may then be cautiously lessened in amount so as to permit sufficient cough to get rid of old clots and other sputa. The invalid should, however, be cautioned against indulging his inclination to cough lest he thereby do harm to the partly healed break in the vessel-wall.

Morphine is less disagreeable than the repeated injection of atropine and is probably more efficient than other agents. Nevertheless it is well to maintain low blood-pressure throughout the aortic system and for this purpose aconite, nitroglycerin and ipecac are all employed. *Aconite* is extensively administered for this purpose and must be ordered in sufficient dosage to keep the pulse soft, full and slow. This treatment has the indorsement of experienced clinicians but to my mind it is not so good as morphine in the manner given above.

Aconite is direct poison to the heart-muscle as well as a sedative to the vaso-motor system of nerves, and lowers vascular tension by virtue of its action on the heart and vessels, but its poisonous action on the myocardium is greater than that on the vaso-motor nerves. Hence, in such doses as are required to keep down intrapulmonary blood-pressure, it is a dangerous remedy to persons already weakened by disease and loss of blood. For these reasons I have never prescribed it in cases of hæmoptysis, although I have had occasion to observe its effects.

Nitroglycerin and allied nitrite compounds are also sometimes prescribed in cases of recurrent hæmoptysis because of their well-known action in reducing arterial tension. It has been especially recommended by Fick for the lessening of high pulmonary blood-pressure as evinced by marked accentuation of the second pulmonic tone. But I must agree with Sticker in the fear that its employment would be more likely to do harm than good. The initial effect of nitroglycerin is acceleration of the heart's action, and it is conceivable that sudden quickening of heart-rate might open a vessel afresh before the secondary dilating effect on the arterioles could appreciably lower the tension in the pulmonic system.

Ipecacuanha is the third and last remedy mentioned as capable of reducing blood-pressure in the aortic and secondarily, therefore, in the pulmonic system. Any substance which occasions nausea occasions slowing and softening of the pulse and hence any member of the group of emetics is capable of producing the same effect as ipecac. Indeed it may be stated *en passant* that this drug taken in a full emetic dose has been employed for the arrest of pulmonary hæmorrhage, but this is altogether too heroic a treatment, especially as we possess other more certain and less disagreeable agents.

There is no valid objection, however, to the use of ipecac for the purpose of preventing pulmonary hæmorrhage since its employment is not dangerous.

Any preparation may be selected, the one requirement being that the remedy be given in such dosage as will keep the patient on the edge of emesis, as it were. So long as the skin is kept warm and moist by the medicine and the pulse soft and full, all is accomplished that is desirable. Should nausea or even vomiting occur, no harm is likely to ensue as the strain of emesis is offset by the beneficial action of the ipecac.

It now remains to consider some other much used remedies, viz., the prolonged administration of ergot either by mouth or under the skin, and astringents as acetate of lead, gallic acid and digitalis. Astringents may be dismissed with the statement that they are utterly worthless in pulmonary hæmorrhage and only do harm by disturbing the stomach.

The consideration of *ergot* may not be so summarily dismissed because it is probably more widely prescribed for hæmoptysis than any other one drug. *Notwithstanding its popularity it is not only irrational for pulmonary bleeding due to erosion or rupture of a pulmonary artery, but it is capable of doing harm.* Ergot raises blood-pressure in the aortic system by contraction of the arterioles through stimulation of the vaso-motor nerves. Whether indirectly through its action on the aortic system or directly on the pulmonary vessels, ergot, therefore, raises blood-pressure in the lungs and defeats the purpose intended.

If hæmoptysis were in every instance due to relaxation and transudation ergot might do good, but when profuse pulmonary bleeding arises in consequence of loss of continuity in the vascular wall, and nature is striving to close the edges of the wound, the attempt to accomplish this must be frustrated by a remedy which, like ergot, raises blood-pressure within that injured vessel. This theoretical consideration restrains me from prescribing ergot in cases of hæmoptysis.

I realize that it is and frequently has been given and apparently with beneficial results. But I cannot help believing that it is in reality a matter of *post hoc* not *propter hoc.* In other words, that if bleeding has ceased after the administration of ergot it has been in spite of and not on account of the drug.

Another very great objection to this medicine lies in the gastric disturbance which it practically always creates. From every standpoint, therefore, I feel compelled to raise my voice in protest against the use of this agent in cases of hæmoptysis.

Digitalis is another therapeutic agent which only very exceptionally finds a place in the treatment of pulmonary hæmorrhage. When bleeding is active, this remedy is too slow in action even were it not actually contraindicated. When a recurrence is apprehended digitalis is inadmissible, nay harmful, because of the increased vigor of cardiac systoles and the augmentation of blood-pressure which it occasions. Aside from the hæmoptysis of cardiac disease the only condition in which I can conceive of its doing good, is when, after the lapse of many days or weeks without serious recurrence, the heart has grown feeble, stasis in the pulmonary vessels ensues and bleeding may be feared in consequence of such passive congestion.

Lastly, in all cases of serious pulmonary hæmorrhage the patient, even though he be a consumptive, must be kept on a *low diet* immediately after the hæmoptysis, and indeed so long as fresh bleeding is apprehended. The food at first should be cold as well as nonstimulating, and in my opinion not confined to fluids so that the amount of blood may not be too rapidly restored to its normal limits. Nature will restore the bulk of the blood without the ingestion of liquids by the sufferer.

When sufficient time has elapsed to warrant an increase in the dietary, even then the patient must cautiously return to his former liberal diet lest pulse tension be too quickly augmented and the vessel be caused to reopen thereby. It is a matter of observation that consumptives are apt to experience a fresh hæmoptysis whenever the dietary has become too liberal. Better, therefore, a light diet and no hæmorrhage than heavy diet and another loss of blood to offset its benefits.

CHAPTER XIII

ACUTE FIBRINOUS PNEUMONIA

Definition.—An acute infection, characterized pathologically by an inflammatory exudate in the lungs and clinically by an abrupt onset, a continued, usually high fever and, in favorable cases, after a limited number of days by a more or less sudden decline of symptoms and return to health with resolution of the inflammatory process within the lungs.

Introductory.—Various terms are applied to this disease but none is quite free from objection. The designation lobar pneumonia was given to it by Laennec because it was noted that in typical cases the inflammatory process involved the whole of a lobe and not groups of lobules. It is now known that the local process may have a lobular as well as a lobar distribution and therefore this term is not scientifically accurate. It has the sanction of usage, however, because agreeing with the facts as observed in most cases, and is the name most commonly given to the disease in this country.

The appellations *croupous and fibrinous* are much employed by the Germans and, owing to the wide influence of German medical thought, are becoming more and more prevalent in other lands. Rokitansky first called it by the former term because of the analogy between the exudate in this disease and that found in the larynx in cases of membranous croup.

Fibrinous pneumonia was the designation chosen by Virchow on account of the richness of the exudate in fibrin, and is probably on the whole the best term that has yet been devised. The affection has also been called pneumonitis by writers like the late Austin Flint, who considered it as a true inflammation of the lungs, and on the other hand acute pneumonic fever, a term which emphasizes the general rather than the local condition. Among the people it is often spoken of as lung fever, or as inflammation of the lungs. The adjectives lobar, croupous and fibrinous will be used interchangeably in this article.

The disease under consideration has existed since the earliest period of medical history, but was not clearly distinguished from other inflammatory processes within the thorax. The only means of diagnosis lay in the study of symptoms, and hence it is not strange that diseases in which pain in the st is a marked clinical feature were confounded. Consequently pleurisy l pneumonia were confused and indeed the latter was thought to have its ting point in inflammation of the pleura. It was not until the beginning nineteenth century, after percussion and its twin sister auscultation given to the profession that a differential bedside diagnosis became possible. It was also not until the last century was well advanced that the pathological anatomy of the disease became well understood and the true relation between it and pleuritis was definitely recognized. Even then it was thought to be essentially an inflammation of the lungs until bacteriological investigation showed it due to the action of micrococci, and a new conception of its nature was entertained. It is now known to be an infective process and the constitutional disturbance a manifestation of bacteræmia, not of inflammatory reaction.

It is still not definitely settled in the minds of all whether pneumonia is a general infection and the lung-involvement a local result or the disease is a local infection and the fever and other constitutional symptoms the result of the local condition. There are a good many facts going to bear out the correctness of the latter theory and many writers of prominence have adopted it (Liebermeister, Andrew H. Smith, Fraenkel).

In favor of the contention that the avenue of infection is through the lungs and that therefore pneumonia is a primary local infection with secondary toxæmia or bacteræmia may be cited the investigations and experiments of Duerck on which Fraenkel lays much stress. He found that the lungs of healthy animals as well as of individuals dead of other diseases than pneumonia contained bacteria of various kinds among which were pneumococci. Furthermore, insufflation of pneumococci into the trachea of rabbits failed to excite pneumonia unless they were accompanied by some irritant, as dust-particles.

Hence Fraenkel concludes that the diplococcus lanceolatus produces the disease of which we are writing only when the germs are unusually virulent or when the natural protection residing in healthy lungs has been overcome by some deleterious influence. Under such conditions a local infective inflammation is set up which is accompanied or speedily followed by general symptoms indicative of pneumococcæmia.

The correctness of the view that pneumonia is a secondary localization of a primary infection of the blood, seems borne out by the experiments of Schultz who produced pneumonia in rabbits by the intravenous injection of pneumococci. Some of Rosenow's blood examinations in cases of pneumococcæmia also appear to support this contention. For instance, he obtained the diplococcus from the blood of five patients before physical signs of pulmonary involvement could be discovered. Furthermore, pneumococci were demonstrated in the blood of individuals who were evidently suffering from some form of septicæmia without the development of pneumonia at any time in the course of their disease. In one of these cases the atrium of infection appears to have been an inflammatory process within the nasal cavity.

The ætiology of pneumonia is still further complicated by the consideration that various micro-organisms, e. g., Friedländer's pneumobacillus, the influenza bacillus, the streptococcus, etc., are capable of producing inflammation of the lungs. The query arises, therefore, are such inflammations to be classed under fibrinous pneumonia in the strict sense or are they similar to this form only in their extent and possibly in their clinical manifestations, whereas in their morbid anatomy they are different?

For example, Friedländer's pneumobacillus produces a form of pneumonia which, in physical signs, cannot be distinguished from ordinary pneumococcus pneumonia. When, however, such a case comes to autopsy it is found that the cut surface yields a stringy mucus so tenacious that it can be drawn out in long strings like chewing-gum. The sputa also contain the same tough stringy mucus. I have been told by Dr. C. S. Williamson, who saw numerous instances of Friedländer's pneumonia in Leipzig, that the presence of such mucus in the sputum and on section of the lung is regarded as so characteristic that on this appearance alone the diagnosis of a Friedländer pneumonia is made.

Fraenkel holds the opinion that genuine fibrinous pneumonia is caused by the diplococcus lanceolatus exclusively, and that when inflammation is produced by other organisms it is not strictly a fibrinous process. He believes, furthermore, that the so-called atypical pneumonias which are often supposed due to some other bacterium than the pneumococcus or the coöperation of this with some other organism, e. g., streptococcus, and are sometimes spoken of as mixed pneumonias, are in reality due to the pneumococcus when viewed from the pathologico-anatomical and bacteriological standpoint.

The pneumococcus is readily killed and replaced by other bacteria, and therefore when cultures made from the lungs after death fail to show this germ one cannot conclude that it was not present as the causative factor. If the cut section shows the characteristic granular appearance, then it is a pneumococcus pneumonia, whereas the inflammatory exudate caused by other organisms is less typically granular and less compact. Weight of evidence seems to support Fraenkel's contention and to indicate that clinical variations in the course and symptoms of acute pneumonia are the result of modifying influences, as age, habits, etc., excepting in those rare cases which, judged by the appearance of sputum and cut surface, are due to the Friedländer bacillus. My treatment of this subject, therefore, will be based on this belief.

Ætiology. Bacteria.—There is still much for bacteriologists to clear up regarding the rôle played by micro-organisms in the causation of pulmonary inflammation. It appears to be settled that a lobar as well as a lobular inflammation may be produced by the action of several different bacteria and, therefore, one cannot assert when speaking in a strict pathological sense that there is but one kind of organism capable of exciting acute pneumonia.

In 1882 Friedländer identified a bacillus which was pathogenic for cats, dogs, guinea-pigs and mice but not for rabbits and which, for a time, was thought to be the cause of pneumonia in man. This latter opinion is now known to be correct, but as already stated this is not the form of pneu-
arily encountered. The so-called Friedländer pneumonia occurs
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In other respects it may closely resemble any ordinary case of
and, excepting for the stringy mucus in the expectoration,
cally differentiated.

r discovered in human saliva a diplococcus which was pathogenic for rabbits. In the same year Talamon obtained this organism from pneumonic lungs, and again, in 1884, it was obtained by Salvioli. In this latter year A. Fraenkel also obtained a diplococcus from hepatized lungs which he cultivated and proved experimentally to possess pathogenic properties. His observations were thereafter confirmed by Weichselbaum and many others; so that Fraenkel's diplococcus lanceolatus soon came to be generally recognized as the cause of acute fibrinous pneumonia.

Indeed, it may be said that doubt on this point no longer exists and when physical findings in an obscure or atypical case are not such as to render clear the nature of the illness it has become a routine method to make smears of the blood or to make cultures and search for the pneumococcus in the belief that if the case is one of fibrinous pneumonia this germ will be found in the blood.

The pathogenic properties of this coccus have also been abundantly attested by its detection in the inflammatory exudates of other organs and tissues than the lungs. Indeed there is scarcely a structure of the body susceptible to invasion in which it has not been found.

Although the diplococcus lanceolatus is now generally accepted as the cause of acute fibrinous pneumonia, still it is not always capable of demonstration in the pneumonic exudate after death. In the instances in which it has not been identified we must conclude that either it was not present originally or, by reason of its easy vulnerability, had been destroyed *in loco.* In other instances it has been found associated with other bacteria, so that we are forced to conclude either that there may be a mixed infection in some cases of pneumonia or that additional micro-organisms gain access and become active in the latter stages of the process (Liebermeister).

On the whole, it may be affirmed that doubt no longer exists concerning the rôle played by the diplococcus lanceolatus in the causation of primary fibrinous pneumonia. The questions that are now engaging the attention of investigators have to do with the conditions favoring infection rather than with the nature of the infective organism. Admirable work has been done along this line under the auspices of the Medical Commission for the Investigation of Acute Respiratory Diseases of the Department of Health of the City of New York, and I shall make free use of the conclusions contained in the first report of this commission.

Viability of the Pneumococcus.—The first question that interests us from a prophylactic standpoint has to do with the length of life of the germ after it leaves the animal body. This was investigated by Francis Carter Wood and the literature of this phase of the subject was reviewed. His summary and conclusions may be reproduced at length without apology.

(1) In moist sputum kept in the dark at room temperatures the average life of the pneumococcus is eleven days, though considerable variations may be noted in different specimens of sputum.

In the same sputum kept at 0° C. the average life of the organism is thirty-five days.

In sputum kept at room temperatures and in a strong light the pneumococcus lives less than five days.

(2) In dried sputum (*a*) in the dark the pneumococcus lives on the average thirty-five days; (*b*) in diffuse light, thirty days; (*c*) in sunlight, less than four hours.

(3) In powdered sputum even when kept in the dark the death of the pneumococcus takes place in from one to four hours. When exposed to sunlight death occurs within an hour.

(4) No important differences were noted in the life of the pneumococcus when dried on glass, tin, or wood. On cloth the life was usually slightly longer than on nonabsorbing surfaces.

(5) Sprayed sputum particles remain in suspension for twenty-four hours, but all masses of a size sufficient to contain bacteria settle at the rate of about 40 cm. per hour.

(6) When sputum containing pneumococci is sprayed the organisms rarely survive for more than an hour, and often die in less time. The substance upon which the particles fall makes but little difference in the life of the organism. On cloth a slight prolongation is occasionally noted, due perhaps to the slow drying.

(7) The mucus of the sputum exerts a destructive action on the pneumococcus.

(8) Exposure of bacterial spray to sunlight while in suspension results in the destruction of the pneumococcus within half an hour.

(9) The conclusions of practical importance which can be drawn from the facts given in this paper are as follows:

A. The life of the pneumococcus in moist sputum is of considerable duration, the average period being less than two weeks unless the material is exposed to direct sunlight. But such sputum does not give off bacteria even when exposed to strong currents of air; it may be considered as innocuous except to persons handling clothes, bedding, etc., which have recently been contaminated. Under ordinary conditions, however, this sputum dries in the course of a few hours or days. The dried masses retain their virulence for a long time, and if deposited on the floor or on the bedding of the patient may be powdered mechanically, as sweeping, dusting, or brushing; the contaminated articles will distribute pneumococci in the air. Fortunately, however, the organisms in the sputum do not remain long in suspension and die off rapidly under the action of light and desiccation. In sunlight or diffuse daylight the bacteria in such powder die within an hour, and in about four hours, if kept in the dark. The danger of infection from powdered sputum may, therefore, be avoided by ample illumination and ventilation of the sick-room in order to destroy or dilute the bacteria, and by the avoidance of dry sweeping or dusting. Articles which may be contaminated
which cannot be cleaned by cloths dampened in a suitable disinfectant
l be removed from the patient's vicinity.

When a person suffering from a pneumococcus infection coughs,
expectorates, or talks, particles of sputum or saliva are expelled from
which may contain virulent pneumococci. Such particles remain
in the air for a number of hours if the ventilation of the room
d. They may be inhaled by persons in the vicinity of the patient

or they may be deposited upon various articles in the room. Whether suspended in the air or dried on surrounding objects, the writer's studies show that they become harmless in a very short time, about an hour and a half being the extreme limit, while many of the pneumococci in the spray perish in a few minutes, especially if exposed to strong light.

In the light of these experiments the risk of infection from the pneumococcus is largely confined to those in direct contact with the person whose excreta contain the organism.

Avenue of Entrance of the Pneumococcus.—So many have been the investigations to determine the channel by which this germ gains access to the human organism that it would be tedious and unprofitable to pass them in review in this work. It will suffice, therefore, to state the conclusions that have been arrived at and are now generally accepted. Netter, who was one of the first to endeavor to answer this question, identified the pneumococcus in the oral secretions of 20 per cent of individuals who had never suffered from pneumonia and in 80 per cent of those who had experienced the disease some time in the past. With the lapse of time, however, this latter number was diminished, but 67 per cent showing the organism in their mouths after five years following their attack. It is especially worthy of note that he was able to obtain the diplococcus from some individuals whose pneumonia had occurred ten, fifteen and even twenty years before.

These results have been confirmed by many other investigators although the figures have varied somewhat. Dr. E. F. Wells, in the Winter of 1904, made smears and cultures from the throats of 135 persons, some of whom had had pneumonia while others had not, although many of the latter were members of households in which the disease had occurred. Of this number 61 or 45 per cent yielded positive results. In some instances Wells identified the pneumococcus in the throat of every member of families in which the disease had recently prevailed.

The latest contribution to the subject of the prevalence of the pneumococcus and allied organisms in the throat-secretions of healthy individuals, comes from the workers under the New York commission already mentioned. They show that an exceedingly rich flora exists in the mouth and pharynx of all persons and that in the great majority, if not in all healthy beings, the diplococcus pneumoniæ may be constantly found. Owing to differences in cultural and other methods employed their findings vary somewhat, but in the main may be said to verify the statement just made. Their researches also proved that other strains exist which are distinguished with difficulty from the genuine pneumococcus. For instance, the streptococcus mucosus of Schottmüller is a frequent inhabitant of the oral cavity and morphologically resembles the pneumonia diplococcus closely.

Park and Williams examined the sputum of 200 individuals for the presence of pneumococci and of this number eighty were classed as normal. In forty of these, or 50 per cent, typical organisms were identified and in eleven the germs were atypical. In the remaining cases no pneumococci were isolated. Loncope and Fox examined the saliva of forty healthy persons and, although failing to demonstrate the presence of pneumococci in from

40 to 50 per cent of the cases, they yet concluded that the organism is an inhabitant of the mouth and throat of most healthy individuals, especially during the Winter months.

Duval and Lewis examined the saliva of twenty-four individuals working in the laboratory of the Boston City Hospital who had never had pneumonia and were in perfect general health. Pneumococci were isolated in every instance, no consideration being given to cultures that did not ferment inulin. Dr. M. W. Hall examined the secretions obtained by a swab from the tonsils and soft palate of thirty-two healthy persons of whom ten were workers in a hospital and the others of miscellaneous outdoor occupations. Typical pneumococci were obtained by growth on poured blood-agar plates in 95 per cent.

Berger in New York found the organism in thirty-nine out of seventy-eight persons and in a second series of 204 examinations of healthy individuals he identified the germ seventy-one times or 34.8 per cent. Berger also found that the lips of drinking-cups as well as of sputum-cups were contaminated by pneumococci and hence he concluded that healthy persons may be infected by contact with pneumonia patients or even with other healthy people who harbor the germs in their mouths.

From the observations thus briefly noted it is a perfectly safe proposition to assert that probably half of all persons carry pneumococci in their throats all the time, while others may have them only in the Winter months or when residing in hospitals or taking charge of pneumonia patients. Others may harbor them only when suffering from pharyngitis or acute coryza.

Nevertheless, in the determination of such facts lies but a partial solution of the problem pertaining to the manner in which the organism gains access to the human system. The normal throat possesses certain natural protecting arrangements against the absorption of such germs as chance to exist within the buccal cavity. Accordingly, the discovery of pneumococci in the throat does not wholly explain the development of a pneumonia.

By some it is assumed that the germ enters the system from the throat by way of the blood or lymphatic vessels. Others believe that the organism in question first reaches the lungs and there, finding a suitable culture medium, grows and thus sets up a systemic infection. As is evident, this at once raises the question as to whether pneumonia is a primary local or a primary general infection.

It may be that the pneumococcus residing in the throat is carried into the lungs and thence into the system, but on the other hand such observations as those of Rosenow appear to show that it may be absorbed from the secretions of an unhealthy nose or throat. The question cannot be satis-
answered at present, therefore. We can only say that the presence
germ in the mouth and throat offers a menace to the health of the
al whenever, in some way, his local or general resistance becomes
l. It is these conditions by which the resistance is impaired that
the predisposing factors.

posing Causes. Individual Susceptibility.—Both clinical obser-
animal experimentation have amply proven that an attack of acute

croupous pneumonia does not confer immunity against a repetition of the infection even for a short time. Indeed, there are numerous published and unpublished instances of individuals who have had the disease many times and even annually. So far from possessing an immunity they appear to possess a marked susceptibility to the disease. Wherein, therefore, lies such a susceptibility or, *per contra*, a relative degree of resistance is a matter for conjecture.

We cannot doubt that some individuals are more susceptible to infection than are others, but in the light of modern facts concerning the causation of most acute disorders, it is a waste of time to talk about inherited or family predisposition. The entire question is resolved into the one of infection, that is, exposure to infection and individual ability to resist infection. Consequently, in the light of our present knowledge we must assume that, aside from the presence of pneumococci in the throat, there is such a thing as individual susceptibility or a lessened resistance which, in some inexplicable manner, depends upon a previous attack, it may be, or upon some other factor not clearly understood.

Exposure.—The influence of cold in the production of pneumonia has always been and still is a much debated question. Before the infectious nature of the disease was established, pneumonia was quite generally believed to be the direct result of taking cold, and figures were adduced to prove such a relationship in a proportion of cases. Thus, Chomel and Grisolle are said by Liebermeister to have found a definite history of exposure to cold in 62 out of 245 and 49 out of 205 cases respectively.

The change in our notions of the nature and cause of pneumonia which has followed the discovery of the pathogenic properties of the diplococcus necessitates an abandonment of the old conception concerning the influence of cold. Writers agree in attributing a certain degree of ætiological influence to exposure, but seem to differ in their estimate of its influence and of the manner in which the cold must be experienced. They are unanimous however in looking upon exposure as but a predisposing cause.

Liebermeister, who appears to consider intense cold, so long as the air is pure, less harmful than the impure air of houses and workshops, cites the immunity of Arctic explorers and of the French soldiers in the Russian campaign of 1812 in corroboration of his contention. Such facts are to be taken, however, as not disproving the influence of exposure but as showing that something else in addition to cold is needful, and this additional factor is the presence of pneumococci in the respiratory passages.

A sudden or prolonged chilling of the body cannot of itself produce pneumonia, but it serves as the trauma which prepares the way for the subsequent action of the germs. In other words, it impairs or destroys the protective power of the bronchial and pulmonary epithelium by occasioning some chemical or biological change in the cells by virtue of which the pneumonia coccus is enabled to find suitable soil for its growth.

Fraenkel's exposition of the manner in which cold works is to my mind very satisfactory. He believes that cold is especially dangerous when the superficial vessels have been previously dilated by severe exertion or by the individual's having been subjected to the relaxing influence of heat inside a building. Under such conditions a sudden transition to cold, as from a draught or upon stepping out into the street, becomes especially dangerous. In support of this theory Fraenkel adduces the well-known frequency of pneumonia among German recruits during the Autumn soon after they have entered upon their Fall maneuvers, and before they have become accustomed to the hardships of army life.

Season.—The influence of season upon the prevalence of this disease is too well established to admit of doubt. Statistics collected with painstaking care by Jurgensen and others have shown conclusively that lobar pneumonia is most frequent in Winter and Spring and least so in late Summer and early Fall. With the advent of cold stormy weather in November cases of pneumonia begin to increase and grow steadily in number as Winter advances, to reach their maximum in March and April. In localities having a sea-climate the disease is most frequent in the early months of the year.

As to the conditions which determine the seasonal differences, authors are not quite united. Hirsch attributed them, with good reason I think, to sudden variations of temperature in the Winter and Spring months rather than to continuously cold weather. Liebermeister suggests that it may be owing, in part at least, to the greater confinement of persons within doors during inclement months. He also calls attention to the remarkable fact that in Germany the prevalence of pneumonia stands in an inverse relation to the precipitation of moisture, being greatest when the rainfall is the least and most infrequent when the rainfall is the most plentiful.

This, it will be noted, corresponds with what was shown by Pettinkofer to be the case as regards typhoid fever and the precipitation of moisture in rain. It is not clear what determines such a relationship, nor whether this exists in all localities with the same distinctness. It may not be an unimportant ætiological factor, for there may be a connection between the dissemination of dust and germs in the atmosphere during dry weather and the spread of acute respiratory diseases.

Climate.—The disease under consideration is met with in all climates, in the tropics as well as in the temperate zone, and in the mountains as well as at the sea level. It cannot be said, therefore, that climate is in itself a direct ætiological factor. Nevertheless, climatic conditions are of indirect influence in that in the temperate regions there are seasonal peculiarities which powerfully affect the prevalence of this disease. Although pneumonia is observed in warm equable climates, it is notoriously more frequent in those countries which in certain months experience abrupt and extreme changes of temperature.

Overexertion.—It has been shown that among the recruits of the German army, pneumonia begins to become frequent when the troops are subjected to the hardships of the Fall maneuvers. As this is at a time when, among civilians, the disease is not very common, Aufrecht is inclined to attribute to strain. The rapid evolutions required of the soldiery necessitate accel- ed and deep breathing, and it is conceivable that the diplococci residing e nasal and oral passages are, under such circumstances, aspirated into the lungs where, under the added influence of exposure, they eventually excite pneumonic inflammation.

Inhalation of Irritating Gases or Smoke.—Under this head comes the inhalation of ether during operations, the fumes of bromine, and nitric or other strong acids, ammonia and even dust. It is a well-known clinical fact that individuals who have been subjected to the inhalation of such chemical irritants are very apt to develop inflammation of the parenchyma of the lungs as well as of the air-passages.

Two queries arise, namely, (1) Is the pneumonia a veritable fibrinous one, or is it a catarrhal inflammation? (2) If it be a true fibrinous pneumonia, can it be attributed to the action of the irritant *per se*? In answer to the first query it may be said that pathologists generally agree with Fraenkel and Aufrecht in the opinion that the pneumonia observed in this class of cases is catarrhal and not fibrinous. In other words, although the clinical signs may appear to indicate a lobar pneumonia as regards the extent of the process, it is in reality a broncho-pneumonia.

Doubtless there are instances of true croupous pneumonia following accidents of the kind now under consideration, but Aufrecht states in no equivocal terms that when such occurs it is attributable to the aspiration of oral secretions or mucus during the state of unconsciousness that is so apt to succeed the inhalation of the irritating fumes. Such is also the view entertained by Fraenkel as regards the so-called ether-pneumonia.

The fumes of mineral acids set up intense inflammation and even œdema of the larynx, trachea and large bronchi. An intense and often widespread bronchitis results, and if the bronchitis extends to the bronchioles it becomes associated with broncho-pneumonia.

The breathing of dust rarely leads to active or acute inflammation but rather to chronic bronchitis. If, however, it induces pneumonia it does so by mechanical injury of the epithelium which lines the air-passages and which is intended to serve as a protective arrangement against the action of harmful germs.

Aufrecht narrates the cases of two young children who were overcome by the inhalation of smoke from a burning feather bed and who developed catarrhal pneumonia. The younger one, aged three years, died and at the autopsy was found to have had catarrhal pneumonia of both lower lobes and of the middle lobe of the right lung.

On the evening of February 1, 1905, a young man, aged seventeen, was admitted to my service in Cook County Hospital who had been carried unconscious from a burning building. His respirations were sixty-eight, his pulse was 160 and his rectal temperature was 97° F. When I saw him on the morning thereafter there was still a strong odor of wood smoke about his person and he was so hoarse as to be unable to speak above a whisper. He was breathing rapidly, about sixty a minute, but otherwise was not suffering. His temperature was approximately 101° F., his pulse was 140 and his chest was filled with numerous fine râles both sibilant and subcrepitant.

Below the angle of the left scapula was a small patch of indefinite dullness over which breath-sounds were feebly bronchial and fine bubbling râles could be heard. The condition was believed to be an intense and extensive inflammation of the air-passages from larynx to small bronchi in consequence of the inhalation of hot air and smoke.

During the succeeding days the patient's condition became gradually more critical. Fever ranged from about 103° F. rectum to 105° F. and even touched 106° F. (Fig. 19), while the respirations remained from forty to sixty and the pulse from 140 to 160. On the sixth day of his illness well-marked dullness was discovered over the greater part of the right lower lobe behind, the breath-sounds here were distinctly bronchial and subcrepitant râles were audible. The next day the patient was delirious and restless, and he died at 11.30 A.M. on Wednesday the seventh day after his exposure to the smoke.

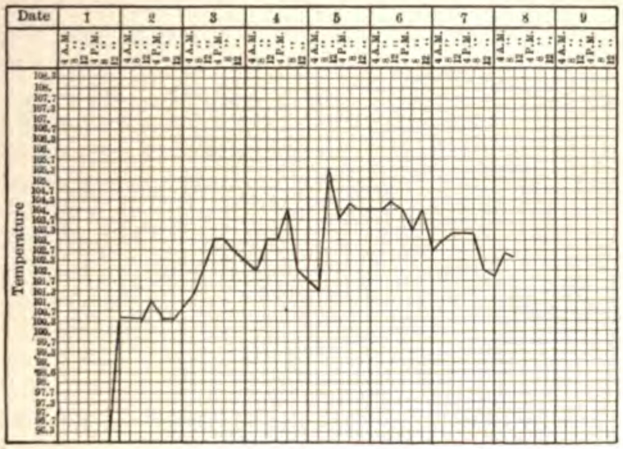


Fig. 19.—Temperature chart in case narrated on page 194.

To judge from physical signs the condition was a pneumonia that had become lobar in extent, yet it was believed to be catarrhal. An autopsy could not be secured and hence the exact nature of the pulmonary changes remained in doubt. It is possible, as there were other pneumonias of true pneumococcus origin in the ward at the same time, that a fibrinous pneumonia had become added to the widespread bronchitis. Such does not seem likely, however, and this case has been thus briefly described for the purpose of bringing out the fact that, in cases of this kind, it is not always capable of clincial demonstration whether the pneumonia be a secondary fibrinous one, or catarrhal.

In connection with this phase of our subject it is interesting and important to note what Fraenkel says concerning the ætiological connection between *partial drowning* and acute pneumonia. He has notes of about a
stances of individuals who, either by accident or intention, narrowly
asphyxia in water and who developed acute pneumonia directly
r. The majority of the cases recovered. Fraenkel explains the
nt production of the acute inflammation by the supposition that
ere carried into the lungs along with the water and that the trauma
onary parenchyma thus occasioned favored the specific action of
coccus.

Acute Diseases.—Leaving out of consideration the pneumonias which sometimes occur in the course of measles and other eruptive diseases of childhood, and which are broncho-pneumonias, it may be stated that inflammation of the lungs may be encountered in certain acute infectious disorders such as influenza and typhoid fever, which bear both clinical and pathological evidence of being fibrinous.

An individual who has been suffering from what was considered only a "slight grippal attack" persists in going to business or exposes himself by going out before his fever and catarrhal symptoms have quite left him. Shortly thereafter, perhaps that very night, he is seized with a chill to which is added a sharp pain in the side and the next day he is manifestly a sick man. The physician who has been summoned recognizes the possible dangers of the case but cannot at once make out a pneumonia.

Some hours or a day later all the signs of lobar pneumonia are present. The sputa are examined and reveal both diplococci and influenza bacilli. The case is regarded as a pneumonia of mixed infection. It is, on the contrary, a true pneumococcus inflammation for which the *grippe* germ has prepared the soil. In the suddenness of its onset, in its clinical features, in its termination and in its morbid anatomical characters it is a true fibrinous inflammation of the lungs.

Such cases are greatly dreaded because of their high mortality, since the pneumonia attacks the individual when already impaired in his vitality by the antecedent influenzal poison.

There are other cases in which the pulmonary inflammation is a true influenza pneumonia, that is, a catarrhal pneumonia of lobular or patchy distribution. Such do not begin in the same manner and do not display the same clinical picture, and if pneumococci are found in the expectoration along with influenza bacilli, these latter greatly preponderate. These cases are also dangerous enough and hence add to the mortality records from *grippe*. Nevertheless, such constitute but a minority of the cases of fatal pneumonia observed whenever an epidemic of influenza is abroad.

Since 1889 *la grippe* has been so frequent and widespread that it seems to have become endemic in many of our large cities, and, since it may undoubtedly act as a predisposing cause of the form of pneumonia of which I am writing, it is largely responsible, in my belief, for the great prevalence and fatality of acute pneumonia in recent years in thickly settled centers of population.

In *typhoid fever* genuine croupous pneumonia is also occasionally seen. Two such instances have fallen under my observation during this past Winter, one of which recovered and the other died. The bronchitis associated with the typhoid is probably a predisposing factor and yet it is remarkable that it so rarely leads to pneumonia. From a differential standpoint it should be borne in mind that hypostatic congestion of the base of one lung may closely simulate a pneumonia.

Chronic Diseases.—Certain chronic affections, notably those of the heart, kidneys and blood-vessels, are well recognized predisposing causes of the so-called *secondary pneumonia*. This complication, if such it may be termed, often sneaks in insidiously and presents a somewhat atypical course and picture, but now and then the pneumonia is quite frank and pronounced. Such an instance will be found narrated subsequently.

It is believed that the chronic congestion of the bronchi, secondary to the cardiac disease, predisposes to the pneumonia by chronic catarrh and desquamation of alveolar epithelium, in consequence of which the soil is prepared for the growth of the pneumococci gaining access to the lungs (Fraenkel).

Chronic tuberculous patients occasionally contract acute croupous pneumonia, but according to figures given by Fraenkel such is relatively very infrequent. He gives some very interesting figures bearing on the relationship between these two diseases. During a period of seven years there were admitted to his service 760 cases of acute croupous pneumonia and of these fifteen or about 2 per cent showed tubercle bacilli in the sputum. When these fifteen cases of combined pneumonia and tuberculosis are compared to the total number of tuberculous patients (3,250) treated in the same period, they are found to constitute but 0.46 per cent of that number. When the total number of pneumonias is compared with the 21,430 admissions from all causes, it is perceived that they constitute 3.5 per cent. In other words, but 0.46 per cent of the tuberculous had pneumonia as against 3.5 per cent of all patients taken together.

Diabetes mellitus is another chronic disease which, by reason of the decreased vital resistance it entails, may be included among the complaints predisposing to acute fibrinous pneumonia. Observations on this point are conflicting, however. According to Fraenkel, Seegen found this form of pulmonary inflammation in nine out of ninety-two *post mortems* on diabetics and Ogle found it twice in fifteen cases. On the other hand, Senator observed but seven instances of pneumonia, four of these fatal, among 700 cases of diabetes, and Frerichs saw only one case of this pulmonary disease in fifty-five autopsies on diabetics.

From Naunyn's observation of the tendency for acute pulmonary gangrene to develop in cases of severe glycosuria, and from the familiar fact that, when tuberculosis of the lungs supervenes in the course of diabetes, the lung affection is likely to be characterized by rapid caseation and excavation, we may conclude that when acute pneumonia develops in a diabetic there is a more than usual liability to rapid destruction of the involved area.

Fraenkel offers the suggestion that the saturation of the tissues with lymph rich in glucose favors necrosis with suppuration and putrescence, whenever the lung is attacked with inflammation. This hypothesis seems to be in accord with Naunyn's observations and to explain the peculiar liability of diabetics to pulmonary complications.

Age.—Pneumonia is more frequent at either extreme than at the middle period of life. All authors are agreed in regarding pneumonia as very frequent in early childhood, and figures showing its frequency as compared with that of middle life or old age will be found under Pneumonia in In- v, in Chapter XIV. Holt collected 500 cases in children below the age teen, 160 from his own practice and 240 from other sources, which show the frequency of this disease in the various years of early childhood. Of these, seventy-six cases or 15 per cent occurred during the first year; 309 or 62 per cent from the second to the sixth year; 104 or 21 per cent from the seventh to the eleventh year; 11 or 2 per cent from the eleventh to the fourteenth year. According to Holt, the greatest susceptibility appears to be from the second to the sixth year and during this period it is most frequent from the third to the fifth year.

Although robust adults are often attacked by this disease it is, according to the last census report, more than twice as common from the forty-fifth to the sixty-fifth year as before the former, while after the age of sixty-five it is still more frequent. The figures given by Osler are as follows: "The death rate in persons from fifteen to forty-five years was 100.05 per 100,000 of population; from forty-five to sixty-five years it was 263.12; and in persons sixty-five years of age and over it was 733.77." These figures are striking and show how powerful an ally has Death in the pneumonia of the aged. When, furthermore, we consider how many old people are said to die of old age who in reality die from this dread disease, the figures given above become still more impressive. Doubtless the real reason for the prevalence of pneumonia at the two extremes of life is to be found in the lessened resistance incident to childhood and old age.

Sex.—Men are more often stricken with pneumonia than are women. This is a fact not only attested by clinical observation but clearly shown by statistics, the census of 1890 giving 33,757 cases among females and 42,739 among the male population. This preponderance of men is doubtless to be attributed to their greater liability to exposure and all those other depressing influences which predispose to the disease. Yet, although the male sex is more frequently attacked, it is stated that the mortality is greater among women (Liebermeister). The reason for this is thought to be found in the inferior resistance of females.

Alcoholism.—There can be no question concerning the predisposing influence of the abuse of alcohol over this disease. This fact is particularly noticeable among the patients in large hospitals. Not only is pneumonia among this class relatively very frequent, but it is strikingly fatal, the largest percentage of deaths from this malady occurring among drunkards. Atypical and asthenic pneumonia undoubtedly occurs among delirium tremens patients far more often than it is recognized.

After all has been said on the predisposing or accessory causes of this disease, there is still much left to be explained. I recently met a gentleman who unhesitatingly attributed his pneumonia to a sudden chilling when in a greatly overheated condition. He had been engaged in some work in a hotel which threw him into a profuse perspiration and while thus overheated he left the building without making a change of clothing. It was in the evening and a cold wind was blowing which, as soon as he emerged from the hotel, caused an almost instantaneous chilling of his body. Not many hours thereafter he came down with his pneumonia which nearly cost him his life.

Another instance of this kind occurred in the person of a young physician whom I attended in his fatal illness. This young man, an interne in Cook County Hospital, had attended to his duties with untiring assiduity and, although of robust physique, had, it is thought, undermined his resistance. One day in March he visited another of the city hospitals and on leaving this institution, presumably much heated by the atmosphere of the operating room, imprudently rode on the front seat of the cable car for a long distance in the face of a cool if not cold wind. That evening at eight o'clock he experienced the chill and pain which ushered in his fatal pneumonia.

It is such instances as the above which probably account in large measure for the greater prevalence of this disease among males than among females, and also for the generally recognized fact that it is more common among men whose occupation subjects them to the vicissitudes of weather. If, added to the effect of exertion, we take into consideration the influence of alcohol and dissipated habits in undermining the health, it is easy to understand the injurious effect of exposure on such individuals. It is reasonable to assume that in many cases. of pneumococcus infection several predisposing factors are at work simultaneously.

Trauma.—Litten has collected such a considerable number of cases with a history of antecedent injury that he holds trauma to be an ætiological factor in 4 per cent. It is not necessary for the injury to be a penetrating one and consequently it is hard to explain how a mere contusion can contribute to the production of a pneumonia. Possibly the resistance of the lungs becomes impaired through injury to the trophic nerves, or a mechanical injury to the pulmonary parenchyma results from the contusion, even though clinical evidence thereof is wanting. At all events, the predisposing influence of trauma must be accepted as well established.

Epidemic Influences.—The consideration of this portion of our subject would not be complete without mention of the epidemic possibilities residing in pneumonia. Medical literature contains numerous accounts of the outbreak of the disease in houses, prisons, hospitals, barracks and villages and its diffusion in so virulent a manner among the inhabitants as to leave but scant room for doubt concerning the epidemic and contagious influence of the malady.

Before the discovery of the specific cause of the disease observers were at a loss to explain epidemics of pneumonia and sought for the conditions favoring the spread of the disease in defective drainage or unsanitary surroundings in general. In the light of modern facts regarding the habitat of the pneumococcus in the secretions of the mouth or throat and in the sputa of pneumonic patients and of the probability of infection of others in close association with the sick person, it is not difficult to understand the development of epidemics whenever the virulence of the germ is intense and other conditions are favorable.

Careful study of reported epidemics, as at Kimberly, e. g., brings out certain highly significant facts. Not only does the exciting cause appear to have been particularly virulent, but the individuals attacked were given to habits and modes of life calculated to further their infection. In many instances they were packed together in a most unhygienic fashion, as in the epidemic of 1878 at the Mohringen Prison when fifty-eight inmates were attacked. In still other instances the infective agent seemed to hang about certain rooms, as in some of the reported epidemics in French and Prussian barracks, or to have found lodgment in some particular ship. Thus, among the crew of the Jeanne D'Arc stationed in the Mediterranean eighty-six were attacked, and upon their removal to other quarters and the transfer of fresh seamen to this ship the disease broke out among the newcomers, showing conclusively that the vessel herself was infected.

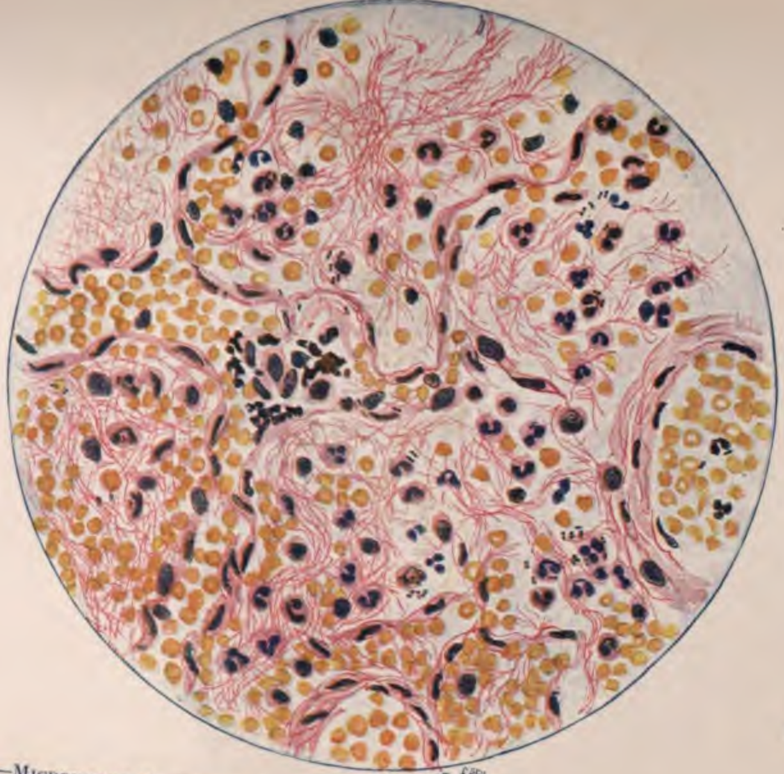
That the malady may be transmitted to individuals through infected clothing and even carried to a distance by a convalescent seems attested by the cases reported by Kühn and recounted by Fraenkel. Kühn contracted pneumonia and soon thereafter certain of his servants who had washed his bed and other clothes. One of these latter, a maid, went home for a visit directly after her recovery and took with her some clothes that had lain in her sick-room. On reaching home she slept with her sister and one week subsequently this latter came down with the disease. These and many other facts attest the communicability of the pneumonic poison and, as will be seen later on, have a direct bearing on prophylaxis.

Morbid Anatomy. Stage of Engorgement.—Pathologists still follow the example of Laennec and divide the pneumonic process into three stages, of which the earliest is that of inflammatory hyperæmia or engorgement. In this stage the affected lobe is congested, of a deep red color, feels firmer and less elastic than normal and the characteristic crepitus is reduced but is still present. On section the cut surface is very moist, and on pressure large quantities of frothy bloody fluid are expressed. Portions cut off float in water, thus showing that the alveoli still contain air. Microscopically (see Plate IV) the capillaries of the alveolar walls are seen much distended with blood; the epithelium of the air cells is swollen and to a large extent detached from the walls. In addition to these desquamated epithelial cells, the alveoli contain red blood-cells and leucocytes in moderate numbers, and usually a considerable amount of blood serum, which is the manifestation of the inflammatory œdema present. The pneumococcus is also usually demonstrable at this stage by cultural methods.

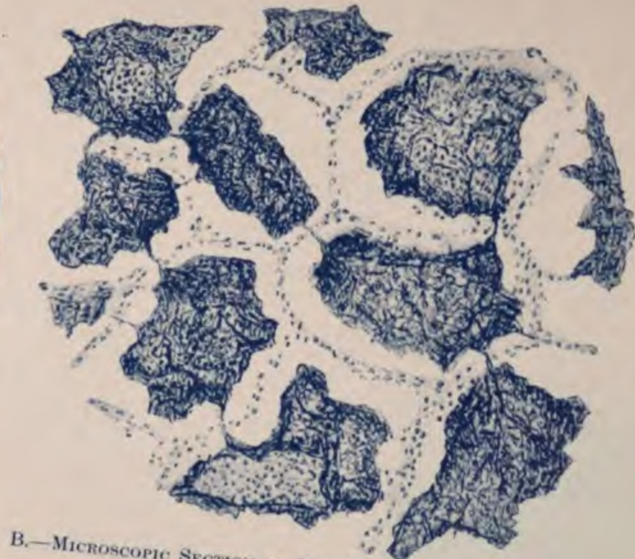
Red Hepatization.—In this stage the affected lobe is firm and airless, and crepitation is entirely absent on pressure. The pleural surface is of a dark, purplish-red color, and is often marked by indentations corresponding to the ribs. Almost invariably when the pneumonic process reaches the surface it is coextensive with a fibrinous or plastic pleuritis of greater or less intensity. The cut section of a lobe in red hepatization is conspicuously dry, and pressure brings fluid only from the largest vessels and bronchi. The surface is noticed to be granular, and on scraping with the edge of the knife the granules come away in the form of small brownish masses, of a pin head size or less. These are, in fact, fibrine molds of the air-cells. The pulmonary tissue is noticeably more friable than normal, and is easily broken down by the pressure of the finger. Pieces cut from the lung in this stage sink in water and are indeed quite airless.

A microscopic section of a red hepatization shows (Plate IV), as its most

PLATE IV



A.—Microscopic Section of Lung of Fibrinous Pneumonia, Stage of Red Hepatization. (Stained with Hæmatoxylin and Eosin.)



B.—Microscopic Section of Red Hepatization. Stained with Weigert's Fibrin Stain. (Fraenkel.)

conspicuous and characteristic feature, a fibrin network containing in its meshes many red blood-cells, a few leucocytes and desquamated epithelial cells. The delicate fibrillar character of this network is seen well in Plate IV, which shows the fibrin stained by the Gram-Weigert method, as was described when considering Plastic Bronchitis. This stain also demonstrates the diplococcus, when it is present and stainable. In spite of the pressure that is exerted by this exudate the capillaries of the alveolar walls remain permeable.

Gray Hepatization.—This stage is perhaps best considered as the beginning of the process of resolution. In it the fibrinous exudate characteristic of the red stage is attacked by vast numbers of polymorphonuclear leucocytes, which, by means of a ferment action analogous to ordinary peptic digestion, bring about the liquefaction of the exudate; this is then, in favorable cases, removed by absorption or expectoration. Gray hepatization is much more commonly seen *post mortem* than is the red form. The gray color is evident on the external surface which is still covered by the fibrinous pleuritic exudate. The lung is still firm and airless and is even more friable than in the red stage. The cut surface (Fig. 20) is now a light pinkish gray mottled with anthracotic pigment in the adult, and is distinctly moist. The knife scrapes from the surface a turbid purulent material which on careful inspection is seen to contain the alveolar plugs, but they are much less distinct than in the second stage. Microscopically the alveolar vessels are usually collapsed and the air-cells are crowded with a dense mass of polymorphonuclear leucocytes. The leucocytes are often so densely massed as to obscure the alveolar structure of the lung (Plate V).

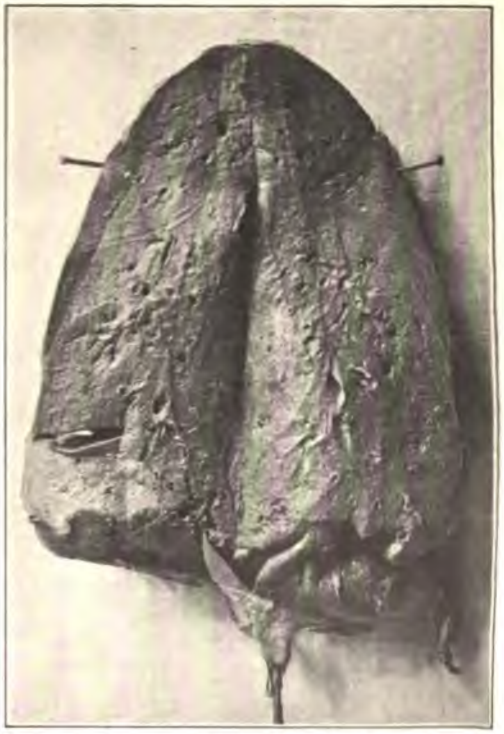
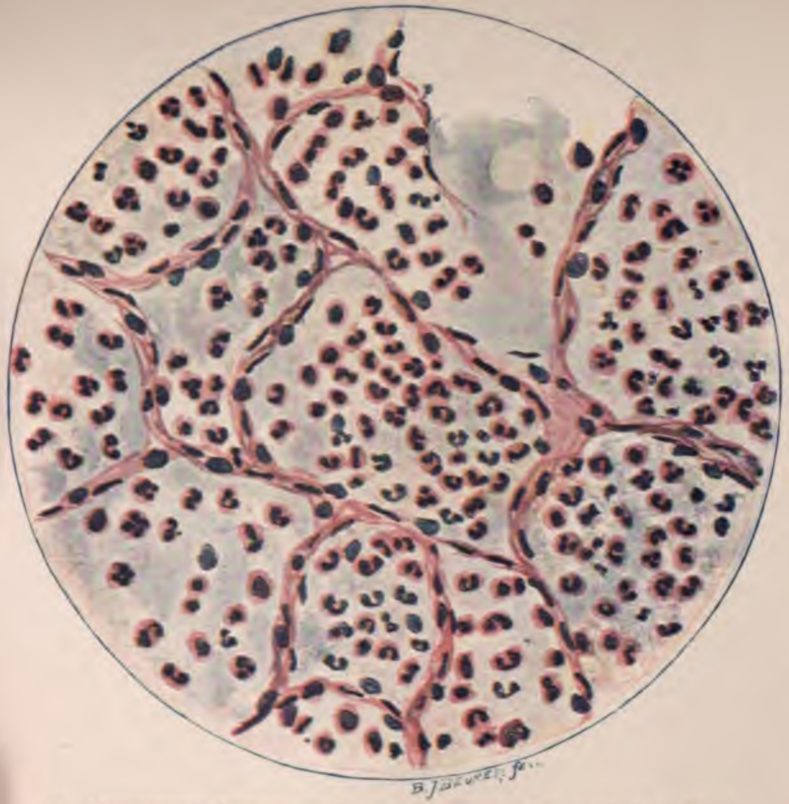


FIG. 20.—Pneumonic lung in stage of gray hepatization.

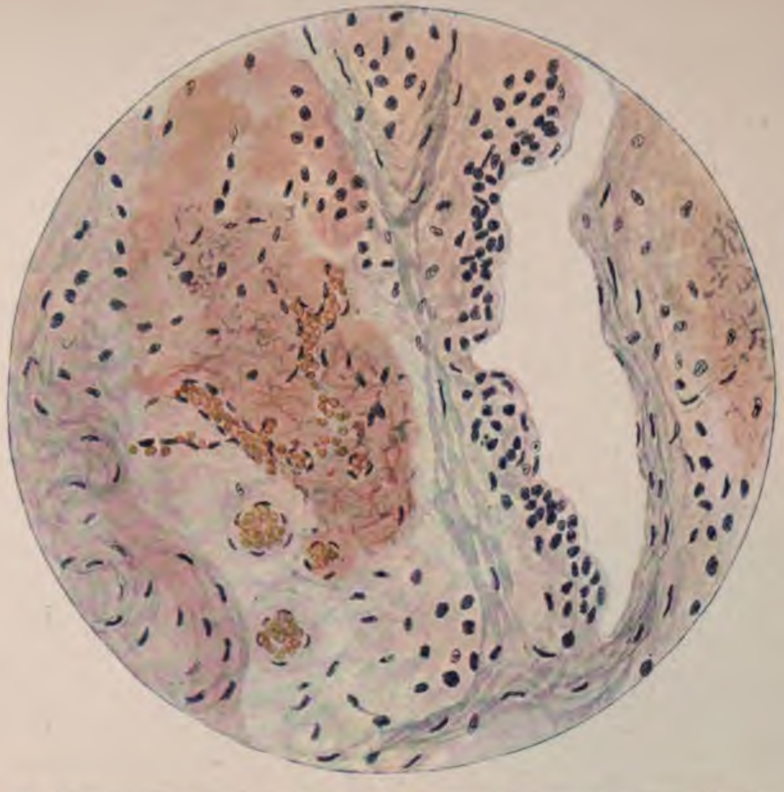
As resolution progresses a larger and larger amount of fluid is formed, and this is evident by increased moisture on gross section and by the presence of coagulated fluid in the cells in the hardened microscopic preparation. In cases going on to recovery this liquefied mass is removed mainly by lymphatic absorption, though to some extent it is probably expectorated. Should death take place the affected lobe may be found in either red or gray hepatization, but much more commonly the latter. Sometimes the peptonizing action of the leucocytes goes so far as to include the lung tissue itself in its action, and thus produces areas of softening or even definite abscess cavities. In other cases, usually when there is present some predisposing cause, such as general debility or diabetes, the affected area may be invaded by the saprophytic organisms of putrefaction, and a focus of pulmonary gangrene results. Such terminations are fortunately rare. A rather more common deviation from the classical course of the disease is the so-called failure of resolution which results in the organization of the exudate. This is usually the result of some condition reducing vitality, such as age, or some secondary infection, as a pericarditis. In this condition the lobe is firm and elastic, crepitates feebly if at all, and on section shows a dry surface with a rather translucent luster. Microscopically, capillary blood-vessels are seen to have invaded the exudate accompanied by leucocytes and young connective tissue cells (Plate V). The affected area is thus transformed into a fibrous mass which, contracting in the course of time, may give rise to much thoracic deformity. (See Chronic Interstitial Pneumonia.)

With regard to the distribution of the pneumonic process among the lobes it is to be noted, first, that the disease shows a marked tendency to involve the whole of any lobe attacked, and usually does so uniformly throughout. It is this peculiarity which has given it the name of Lobar Pneumonia. In considerably more than half of the cases one lobe only is involved, lower lobes more commonly than upper lobes and the right side more often than the left (Chomel, Grisolle, Andral). Juergensen's statistics of 16,600 cases in adults show the right lung attacked in 53.1 per cent, the left in 36.5 per cent, and both lungs in 10.4 per cent. Of Fraenkel's 830 cases, the right lung was involved in 451 or 54.4 per cent, the left in 256 or 30.9 per cent, and both in 121 or 14.6 per cent, while centric pneumonia was observed in fourteen or 1.7 per cent. In 136 cases the upper lobe was affected 16.4 per cent. Fraenkel states also that of seventy-nine cases examined *post mortem* by Koschella the right lung was involved forty-five times, the left twenty-two and both thirty times. Fraenkel finds an explanation for the more frequent involvement of the right lung in the greater caliber of the right bronchus and its more nearly vertical line of descent, pointing out in particular that the branch to the lower lobe continues in nearly the same direction as the main stem. He also looks upon this arrangement as a further proof that pneumonia is a local infection. According to Aufrecht, Grisolle's figures are the following: right lung 166, left 97, both lungs 17, total 280 cases. Huss found the distribution as follows: 2,616 cases, right lung 1,398, left 834, both lungs 384. Stated in percentages Huss's statistics agree closely with Fraenkel's, namely, 53 per cent for the right, 32 per cent for the left and 15 per cent for both lungs. The annexed table showing the individual involvement of the several lobes, and arranged according to age and sex, is taken from Aufrecht's elaborate contribution to the subject of pneumonia in Nothnagel's System, and is so excellent that no apology is made for its introduction here.

PLATE V



A.—Microscopic Section of Lung of Fibrinous Pneumonia in Stage of Gray Hepatization. (Stained with Hæmatoxylin and Eosin.)



B.—Microscopic Section of Lung of Fibrinous Pneumonia Showing Organization of Exudate in Case of Failure of Resolution. (Stained with Hæmatoxylin and Eosin.)

Table Showing the Involvement of the Individual Lobes in 1,501 Cases of Pneumonia

Age (years).	R. L.		R. U.		Middle.		R. Whole.		R. 2 Lobes.		L. L.		L. U.		L. Whole.		Both Lungs.		Total.	
	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.
0– 5....	4	2	5	2	0	0	6	5	3	1	3	7	3	1	6	2	5	2	35	22
	6		7		0		11		4		10		4		8		7		57	
5–10....	3	4	9	6	0	0	0	1	0	0	7	9	2	4	0	0	1	0	22	24
	7		15		0		1		0		16		6		0		1		46	
10–20....	56	19	18	8	9	2	22	6	19	0	83	23	3	4	17	4	29	11	256	77
	75		26		11		28		19		106		7		21		40		333	
20–30....	91	15	31	3	11	2	41	7	34	1	81	16	12	1	23	6	48	14	372	65
	106		34		13		48		35		97		13		29		62		437	
30–40....	57	6	17	5	7	0	16	5	28	1	56	5	13	0	15	2	20	6	229	30
	63		22		7		21		29		61		13		17		26		259	
40–50....	42	4	15	2	1	0	16	2	10	1	42	5	6	1	13	3	27	5	172	23
	46		17		1		18		11		47		7		16		32		195	
50–60....	13	5	11	0	2	0	9	4	5	2	16	1	4	0	1	2	8	0	69	14
	18		11		2		13		7		17		4		3		8		83	
Over 60..	15	4	8	5	0	0	11	0	4	3	16	3	1	2	4	2	9	4	68	23
	19		13		0		11		7		19		3		6		13		91	
Total..	281	59	114	31	30	4	121	30	103	9	304	69	44	13	79	21	147	42	1,223	278
	340		145		34		151		112		373		57		100		189		1,501	
Per cent..	22.6		9.6		2.3		10.0		7.5		24.8		3.9		6.7		12.6		100	

While, as a rule, the whole of a lobe is affected at once, this is not always the case, and at times the consolidation may be limited to the central portion of a lobe and only reach the pleural surface late if at all. Such a condition is called a centric pneumonia and gives rise often to difficulty of diagnosis on account of anomalous physical signs. The same is also true of the so-called massive pneumonia, in which the consolidation involves the bronchi as well as the air-cells and all access of air to the lobe is shut off.

The pleurisy overlying a pneumonic lobe is practically universal except in centric pneumonia. It is usually a fibrinous pleuritis with little if any serous exudation, though the latter may in some instances predominate. Furthermore, especially in children, the pleurisy is apt to become purulent and result in empyema. This is comparatively infrequent in the adult.

Secondary infections of other organs and serous membranes with the pneumococcus or other pyogenic organisms may occur as complications. Pericarditis is not rare and is usually associated with pneumonia of the left side or with double pneumonia. In such a case it seems as though the infection had passed from one lung to the other by means of the pericardium. Endocarditis occurs with varying frequency, Osler's figures of sixteen in 100 autopsies being considerably above the average experience. When it does occur, the endocarditis of pneumonia is more often of the malignant than of the benign type. Meningitis is a rare complication but may be a true pneumococcus inflammation. The usual degenerations of any febrile condition are found in heart, liver, kidneys and spleen. The latter may be much enlarged.

CHAPTER XIV

ACUTE FIBRINOUS PNEUMONIA—*Continued*

Symptoms. General Clinical Picture.—Although not exclusively confined to persons in the middle period of life, typical lobar pneumonia is certainly seen most frequently in robust young adults. So striking is the contrast between the pneumonia of such individuals and that of children and the aged that it may be characterized as the pneumonia of the vigorous young man. The clinical picture is so unlike that of any other disease that when once seen it cannot fail of recognition again. It is scarcely to be wondered at, if old practitioners commit themselves to its diagnosis from the symptoms alone without examination of the chest.

Usually without warning, the individual is seized with a sharp chill that lasts from twenty to thirty minutes. At the same time he feels acutely ill and as the rigor subsides passes into a burning fever which in the course of a few hours mounts up to 103° or 104° F. With the disappearance of the chill he becomes conscious of pain in his side so severe as to make him hold his chest as immobile as possible. His breathing now becomes accelerated and shallow, and expiration is accompanied by a moan or grunt. He lies on the affected side so as to restrict its respiratory motion and endeavors to restrain cough which by this time has become a very troublesome symptom.

If seen on the second or third day when the illness is at its height, the patient presents a characteristic appearance. He lies turned partly to one side, usually the right, breathing rapidly and with an expiratory groan, his eyes bright, face flushed, sometimes cyanotic, and often a dark red spot on one cheek plainly noticeable in spite of the generally heightened color of the countenance.

The skin is hot and perspiring, the pulse rapid, full and bounding, while, at short intervals, the sufferer gives a cough which, because of the pain attending it, is suppressed so far as possible. The cough results in the expulsion of a scanty amount of sticky reddish sputum, so viscid indeed that it often has to be wiped from the lips. Examined more closely in the sputum-cup it is seen to be of a rusty hue, so tenacious as to adhere to the bottom of the cup and quite apt to be associated with more or less frothy serum. The cessation of the act of coughing is attended with a moan of suffering and an increased secretion of sweat.

The patient seems glad to lie still and reluctant to talk, which, as a matter of fact, is difficult on account of his dyspnœa. He has no desire for food, and although his mouth and tongue are dry does not appear to care for water. The urine is scanty and dark colored and on chemical examination is found to possess certain peculiarities that will be described later on. The bowels are generally somewhat confined and often there is considerable distention of the abdomen.

Headache may be severe and sleep impossible on account of the frequent painful cough. In some cases insomnia is pronounced and is then owing to the effect of the toxins on the cerebrum. The intelligence is usually clear but if the fever is high there may be delirium.

Examination of the chest at this time discloses in one lung, most often the right lower lobe, signs of consolidation, viz., dullness and bronchial breathing. There may also be heard the characteristic crepitant râle or fine bubbling râles of associated bronchitis, either limited to the affected lung or more marked there. It is not uncommon also to detect friction from an accompanying pleuritis, but at the height of red hepatization the intensely tubular breath-sounds may be all that is detected on auscultation. The right heart is dilated and the pulmonic second sound is loudly accentuated.

In this condition the patient remains for a variable number of days, five to seven or nine, when a sudden and pronounced change in symptoms takes place, known as the crisis. This is characterized by a more or less rapid fall of body temperature to normal or below. At the same time the patient breaks into a warm, often profuse, perspiration, and, feeling relieved, sinks into a refreshing sleep.

When, after a few hours of grateful slumber he awakes, he is ready for nourishment. His respirations are still accelerated, but cough and expectoration are both easier. To the casual observer the patient is manifestly better than a few hours before and plainly on the road to recovery. In most cases the defervescence is persistent and convalescence proceeds uninterruptedly and rapidly until, at the end of another week or ten days, the patient is able to sit up or even to walk about.

So striking indeed is the contrast between his condition now and that of a week earlier that it seems like recalling a nightmare to think back on his suffering and danger of a few short days before. Perhaps no other acute disease manifests such sudden and pronounced transitions from robust health to dangerous illness and again in a few days from doubtful recovery and distress to a state of comparative comfort and freedom from peril.

Unfortunately all cases do not terminate thus favorably but are unexpectedly cut short in some one of the following ways. In exceptional instances the patient who appears doing well dies with such suddenness as to suggest the formation of a heart-clot, or he begins to display fine crackling râles which increase until pulmonary œdema is recognized and ends the struggle. In other cases the pulse becomes more and more rapid, at last thready, cyanosis deepens to an intense purplish hue, and death occurs through cardiac asthenia. In some the intellect remains clear to the last but in others delirium or coma marks the closing hours.

In still other cases there is no sudden change for the worse but the disease displays malignancy from almost the very first. The pulse soon exceeds 120 and the patient shows every indication of profound toxæmia. The chest displays the coarse, moist râles of acute bronchitis even in the lung that is free from pneumonia. These râles soon obscure the heart and breath-sounds and may even be audible a considerable distance from the patient's mouth. If the sufferer can be induced to suspend breathing for an instant the pulmonic second tone is found extremely weak and even muffled.

The mind may still be unclouded and the sensibility undiminished as shown by the frequent but ineffectual cough. Yet, in most cases, more or less stupor supervenes and renders the patient insensible to the collection of mucus and serum in his air-tubes. The individual is plainly far worse with each successive hour. At length he answers questions reluctantly and imperfectly, seems indifferent to his condition and asks for neither drink nor food.

In still other instances restlessness and evidence of distress are apparent and make it necessary to have the patient supported in a high position that he may breathe with more ease. The distended abdomen is markedly tympanitic and impedes inspiration. The cyanosis at first limited to extremities and lips begins to spread and deepen until at length it gives the skin a generally livid hue. The pulse runs from 140 to 150, is small and of markedly low tension and may even intermit occasionally. The feebleness of the pulmonic second tone gives further evidence of the failing heart-power.

In other cases the surface of the body grows cold and may be covered by a clammy sweat, a condition which together with the weak, small, rapid pulse indicates the much-dreaded collapse from which the patient rarely recovers.

After the lapse of a few hours more the disastrous effects of the toxæmia are still more conspicuous. The skin is everywhere livid and the face looks sunken and pinched while the expression is listless. The extremities are cold and dry like those of a corpse or are covered by a profuse and cold or merely clammy sweat.

The pulse is very thready and rapid. Mucous râles fill the entire chest and rattle in the trachea. Cough is no longer heard by the attendants, urine is passed involuntarily and the patient tends to sink lower and lower into the bed. At length the abdomen becomes retracted, the Hippocratic facies is apparent and a few hours later death mercifully terminates the unequal struggle.

The subjoined case illustrates well the clinical picture of a typical primary pneumonia in a young adult. M. S., age seventeen, stable boy, was admitted to my service in Cook County Hospital January 15th, 1905, with the history of his illness having begun suddenly four days previously. He had been taken with a severe chill after which he became very hot. To these symptoms were added sharp pain in the left side, cough which at first was dry but afterwards resulted in a bloody expectoration. He was short of breath and was delirious part of the time.

Physical Examination.—A colored boy of good size and well developed, tongue heavily coated, eyes bright, heart of normal size and the sounds clear, pulmonary resonance impaired below left scapula and over both lungs a few dry râles, but no distinct signs of consolidation; abdomen and other parts all negative.

The temperature was 103.6° F., pulse 116, and respirations thirty-two; urine analysis showed color brick red, reaction acid, sp. gr. 1.023, a distinct trace of albumin, sugar absent, many casts, chlorides not estimated. Leucocytes at this time were not counted, but two days later they were 19,000. The Chart in Fig. 21 shows the temperature for the next six days until the crisis.

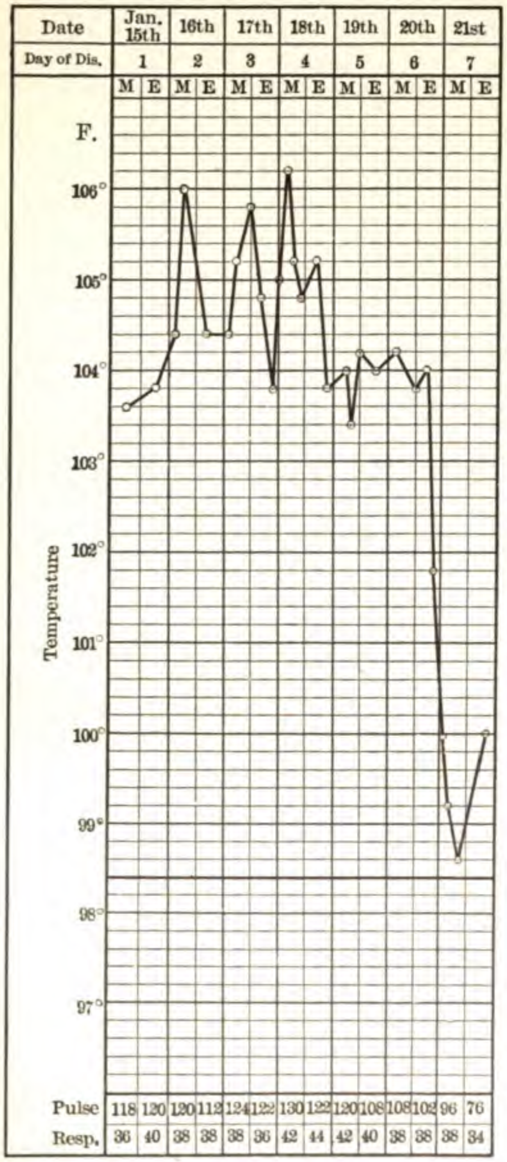


Fig. 21.—Chart showing temperature in case of M. S. narrated on page 207.

On the morning of January 16th dullness, bronchial breathing and here and there a bunch of crepitant râles were present in the left back from apex to base, and on the evening of that same day herpes appeared on the right side of the mouth.

On the 17th it was recorded that patient had slept about five hours but was restless and complained of severe pain in the left side. On the 18th it was further recorded that he had slept most of the night, but was slightly irrational at times and showed some muscular twitchings of the right side of the body. At my visit on that morning I found the boy lying partly turned to left side, very dyspnœic and grunting with each expiration. On examination the entire left lung yielded marked dullness and distinct bronchial respiration, and it was evident that there had been an extension of the process to the entire upper lobe. The temperature was 105.2° F., respirations forty and pulse 128, but as this last was of good quality and the second pulmonic sound clear and ringing no special uneasiness was felt concerning the patient's condition. As nearly as could be determined he had entered the seventh day and hope was entertained that the crisis might occur shortly.

On the next two days the general condition remained about the same except that he was restless and quite delirious, the delirium being that of exhaustion, as is not infrequent shortly before the crisis. Although cough was frequent and breathing labored, still the boy managed to get several hours of sleep. In the evening of the 20th, on what was probably the beginning of the tenth day of the illness, the temperature fell to 101.8° F., and on the morning of the 21st day of month sank to normal.

The patient now expressed himself as comfortable and was entirely rational. Râles of resolution were audible over the involved areas and the percussion-note was becoming less dull and of a slightly tympanitic quality. Convalescence proceeded uninterruptedly and in another week the boy was up and about the ward. Then occurred another sudden elevation of temperature to 102° F. and fear of a relapse was entertained. Examination of the chest was negative however and the following day the temperature had again fallen to normal. To what this sudden flurry was owing could not be ascertained, and February 1st the boy left the hospital fully recovered. It is interesting to note that this case was one of several from the same house.

Unfortunately all cases of sthenic pneumonia do not terminate so happily, even when occurring in young adults, as is shown by the illness of a promising young interne at Cook County Hospital in the Winter of 1904. The pneumonia set in stormily and ran a high fever from the first. Pain, rapidity of breathing, distressing cough, rusty sputa, hot perspiring skin and a full bounding pulse of about 110 being the features of the case, in themselves not at all peculiar or especially alarming. The infiltration was limited to the right lower and middle lobes.

On the third day his condition indicated a profound bacteræmia, although the sensorium was not disturbed nor was anyone able to fasten upon any particular symptom that rendered us uneasy. On the morning of the fourth day, however, his cyanosis had grown more pronounced and his heart-action was more rapid. The young man looked decidedly worse. That evening he complained of increased pain in the right side, and respirations became more rapid and more superficial. He did not sleep but seemed abnormally bright and alert, although not inclined to talk or else restraining himself from conversation because desirous of husbanding his strength.

Monday morning his pulse was still more frequent, about 140, as I now recall it, and plainly smaller and weaker. Cyanosis had deepened materially, the right heart had dilated appreciably as indicated by percussion, the pulmonic second sound was distinctly muffled and some coarse moist râles were present in the front of the chest. The abdomen was tympanitic. It was plain that the patient was failing, and in addition to energetic stimulation venesection was ordered.

During the evening of that day active delirium set in suddenly and the patient had to be restrained. It yielded however to an ice rub and a small hypodermic of morphine. Nevertheless, the general and cardiac condition was rapidly growing worse. Toward morning delirium again occurred, symptoms of approaching collapse appeared, fine crackling râles became audible throughout both lungs and the patient, now wholly unconscious, passed away at 7.30 on the morning of the sixth day of his illness.

The following case of secondary pneumonia, taken from my case records, not quite classical in all its features, some of which are modified by treatment, is yet sufficiently typical to serve as a good example of the disease. A man of forty-eight, having a chronic mitral lesion, but still in unusually good health for him, went to business on Saturday, August 20th, and did a hard day's work.

On returning home to dinner in the evening he complained of indigestion, declared he felt sick and took calomel. As he was not better Sunday morning his family physician, a thoroughly competent man, was summoned. Not being able to discover anything more than intestinal indigestion he prescribed more calomel and citrate of magnesia. That day the patient remained in bed, but that night at about two o'clock his wife, perceiving that he felt feverish, took his temperature and found it was 104.2° F. At this time he complained of a dull pain beneath the left shoulder blade, but Monday morning spoke of it to his physician as being in the region of the left shoulder. The lungs were carefully gone over with purely negative results and the case was regarded as one of intestinal toxæmia, especially as the abdomen was quite tympanitic.

Tuesday forenoon I was asked to see him with his family doctor, owing to great irregularity of the pulse and to my having previously treated his heart lesion. The patient was perfectly clear mentally and did not feel especially bad. His temperature was between 102° and 103° F., his abdomen was markedly tympanitic and his pulse was about 120, but very arrhythmic. His respirations were not apparently accelerated and he had no cough that attracted notice. He mentioned an occasional slight pain in the left shoulder and chest just outside the heart. The lungs were therefore carefully examined, but nothing definite was discovered. Nevertheless, I thought I detected with one or two inspirations what seemed like a faint rub low down at the left not far from the apex of the heart.

Although fearful of something more than intestinal toxæmia I was yet unable to fix on anything in particular, but recommended digitalis to sustain the pulse. Wednesday morning, the fourth day of the disease, his temperature was still 102° F., and the pulse was so weak, irregular and rapid and the man was so nervous, apparently because of a poor night, that a quarter of a grain of morphine was very appropriately administered hypodermically.

At three o'clock P.M. that same day his wife, not being able to get hold of the attending physician telephoned to me that her husband was breathing very peculiarly and his pulse was very weak. Examined an hour later the patient presented the following condition: temperature 103.6° F., respirations forty-four, the pulse so rapid, feeble and irregular that its rate could not be determined, and at the left base behind moderate dullness with tympany, semibronchial breathing and some fine moist but not typical crepitant râles. Below the right scapula was also a small patch of impairment with fine bubbling. The case was plainly one of acute pneumonia, complicated by a chronic mitral affection, and steps were at once taken to sustain the heart by digitalis, strychnine and caffeine, all hypodermically.

Thursday morning the condition was about in *statu quo* with the exception that fluid blood-tinged sputum in small quantities was occasionally expectorated. Cough was however not frequent nor severe, and the patient was perfectly clear and not suffering much distress. The urine at this time showed an almost complete disappearance of the chlorides, in other respects negative.

That same afternoon, however, his condition had grown alarmingly worse. His color was considerably cyanotic, his face looked gray and sunken, his respirations were forty-nine, his pulse scarcely appreciable, but the extremely irregular heart-beats were, as nearly as could be counted, 160. The extreme meteorism was looked upon as of especially bad import, and it was predicted that he could not survive through the night. Frequent stimulation with hypodermics of camphorated oil, and with aromatic spirits of ammonia by mouth was at once begun and kept up until one o'clock Friday morning. At that time the patient rallied in a surprising manner, his pulse became steadier, stronger and somewhat slower, he broke out into a tremendous sweat and turning over on to his left side he went to sleep.

At my visit Friday forenoon his rectal temperature was 101.4° F., he was entirely rational and, although heart-sounds were exceedingly rapid and feeble, his condition appeared decidedly improved over that of the evening previous. Toward noon however he complained bitterly of nausea, and to prevent vomiting which it was thought might prove fatal, he received a small dose of morphine under the skin. This put him to sleep for a while, but at three P.M. he had a sudden sinking spell when he grew almost livid, clutched his throat, raised himself in bed and declared he was strangling. A prompt injection of camphor speedily brought him out of this attack, and when I saw him an hour later he was comfortable but still fighting for life.

His color was quite cyanotic, his extremities were hot, however, except the hands, the pulse was very bad, he lay partly turned to the left, his body vibrated synchronously with his rapid laborious respirations and his arms twitched frequently. He was conscious, but now and then inclined to wander slightly. In this state receiving still energetic and frequent stimulation he remained until about two o'clock Saturday morning, the seventh day of his illness. Temperature had again risen to nearly 104° F. and he was not perspiring much. The skin was neither cold nor hot and his kidneys had been acting fairly well in spite of the large amounts of camphor he had received. He slept at intervals and was not very restless.

Suddenly, somewhere about three o'clock A.M. he grew livid, began to rattle in the throat, sprang up in bed, clutched at the air frantically and seemed in danger of immediate dissolution. Camphor was injected, as well as a small dose of morphine, and that not allaying the attack his physician threw under the skin a fiftieth of atropine. The effect was magical, for almost immediately his color changed, his face filled out, his rattling ceased, his skin grew warm, his restlessness passed and, turning over, he fell asleep.

Toward noon of Saturday another of these alarming spells came on and was promptly allayed as before. A third one took place in the middle of the afternoon and was again arrested by atropine. The increasing scantiness of urine necessitated a discontinuance of the camphor early in the day and this may have accounted somewhat for the frequency of these attacks of impending pulmonary œdema, as they seemed to be. He had, however, been getting carbonate of ammonia and the injections of digitalis, strychnine and caffeine as before. When I called at 5.30 P.M. he was lying on his left side breathing laboriously; he had been sleeping at intervals and had been alternately mentally clear and delirious.

His lungs were free from râles, probably in consequence of the atropine, and the resonant portions seemed rather more resonant than on Friday, but the left lower lobe was still so solid that the heart-tones could be plainly heard behind.

It was realized that his heart was only kept going by the tremendous stimulation or whipping it had received and that it could not maintain its pace of 180 or 190 for long. Suddenly another attack of threatening asystolism occurred and the patient looked as if he would die in a few minutes. Camphor and other stimulants and atropine were quickly administered, but the pulse did not respond although the sufferer grew quiet and appeared to sleep. At this time his color was purple, his body was covered by a cold sweat, his respirations were thirty-eight to forty and extremely shallow, but were free from rattling on account of the atropine.

In this moribund condition he lay for nearly three hours, yet waking up partially when addressed by his wife. At length, however, he ceased to hear her endearing words and when I came away at 9.30 P.M. was to all intents and purposes lifeless, but still breathing rapidly and very superficially while his poor tired heart still kept on feebly fluttering in its vain attempt to sustain life.

The struggle was the most protracted I have ever seen and did not cease until 12.30 A.M. Needless to add, all stimulants were withheld from 7.30 P.M. on. In conclusion the only excuse for so detailed a narration of this case is its instructive portrayal of one of the modes in which this truly terrible disease slays its victims.

Analysis of Symptoms. Prodromata.—As a rule croupous pneumonia sets in abruptly while the individual is in apparently perfect health. Exceptionally, in about 25 per cent of cases (Grisolle) there are prodromal symptoms which last from a few days to even four weeks (Traube). These are loss of appetite, a feeling of *malaise*, backache, headache, pains in the muscles or joints, digestive disturbances and bronchial catarrh. Grisolle is said to have noted bronchitis in nineteen out of his fifty cases in which prodromata were observed.

Although I have no figures showing the frequency with which such symptoms have preceded the actual onset of the disease in my cases, I yet know that among the pneumonia patients admitted to Cook County Hospital a history of prodromal symptoms is very common. In a private case seen in September, 1903 the patient, an elderly man, declared that for four or five days preceding his initial pain in the side he had felt dreadfully tired and thought he had been slightly feverish because on going out into the evening air he had felt chilly.

The insidious onset and prodromata are well shown by the case of Mr. L., a merchant of forty-five, who began to complain on Friday, Jan. 4th of pains in his extremities and in his body generally and felt so cold that he hugged the fire all day. He slept that night, however, and next day kept about, although his aching pains, shiverings and hot flashes continued. That night he was able to sleep, but the following morning, Sunday, he did not get up. The pain had rather left the extremities and concentrated itself in his chest, and he had fever. The family physician, Dr. Morton, was summoned and found that the patient had a temperature of $103\frac{2}{5}$° F.; he pronounced the attack the *grippe* of which there were a good many cases in the city at that time.

For the next three days the temperature remained in the neighborhood of 103° F. and the patient perspired profusely. On Tuesday, the fifth day of the illness, he began to cough, which occasioned much pain in the left side, and raised some mucous sputum. During the next three days the mouth temperature ranged at about 101° F., the patient grew extremely nervous and weak, took nourishment with reluctance, and in the night became mildly delirious. On Friday afternoon, the eighth day of his illness, I saw the patient, in consultation with Dr. Morton, who said he had discovered signs of pneumonia two days before.

The patient was in bed, looking not greatly distressed but feeling much prostrated. The skin was moderately hot and moist, temperature in axilla 101° F., pulse 122, weak but regular, respirations thirty, very little cough. Percussion detected pronounced dullness with great increase of resistance at the apex of the left lung, while at the base the dullness was somewhat less pronounced.

Over the area of marked dullness the breath-sounds were feebly tubular without râles, while over the base the louder bronchial respiration was accompanied here and there, especially around the edges, by fine crepitant râles limited to the end of inspiration. The voice-sounds were distinct but feeble.

The heart was in its normal position and its sounds were clear. The abdomen was tympanitic; the spleen was not palpable; but the liver could be felt a short distance below the lower costal margin. The urine was scanty, 1.028, showed a trace of albumin but no sugar, the chlorides were 4 per cent, blood and both hyaline and granular casts were present. The sputum expectorated during the examination was rusty and sticky, and under the microscope showed mixed pus-cocci; influenza bacilli and numerous pneumococci.

Rigor.—This is an initial symptom of such frequency and distinctiveness as to have formerly been responsible for the belief that pneumonia was the direct consequence of taking cold. When, however, one comes to look up statistics on this subject he is likely to be surprised by the differences found in such figures. Thus Grisolle observed rigor in 145 out of 182 cases, or 78.6 per cent; Louis, sixty-one out of seventy-nine cases, or 78.5 per cent;

Juergensen, sixty out of 100 cases, or 60 per cent; Fraenkel, 184 out of 341 cases, or 53.6 per cent; Morhardt, 41.7 per cent; Menge, 57.6 per cent; Fismer, 38.3 per cent; Stortz, 31.3 per cent.

This chill is, of course, but a symptom of the infection and hence it is not peculiar to pneumonia. Yet it may be safely said, I think, that it is rarely so pronounced in any other acute disease. It consists not of a repetition of chilly sensations, but of a single chill which varies in different individuals as regards intensity and duration. In the aged it may be no more than an ill-defined sense of chilliness or slight shivering, as noted by me in a woman of seventy-nine years, but in the young adult at least it is as a rule a severe rigor, the patient feeling as if chilled to the bone and actually unable to keep from shaking or prevent his teeth from chattering. The duration is usually twenty or thirty minutes, but it may persist for two, three or even nine hours (Lebert).

Vomiting, Convulsions or Coma.—These are seldom, if ever, seen in the pneumonia of a healthy young adult but are not at all uncommon among young children, and are now and then found among the aged. If due care be not observed they may mislead by directing attention from the lungs to the brain.

Pain.—This appears to be a more common symptom than is rigor, having been absent in but twenty-nine out of 201 cases of pneumonia analyzed by Grisolle. It not infrequently ushers in the attack, but when such is not the case it appears early, within the first twelve hours in the majority of instances, and may accompany or even precede the chill (Wilson Fox). The pain is generally acute, not rarely so intense as to be truly agonizing, and is usually located in the affected side over the lobe involved. Since the pneumonia most often invades a lower lobe the pain is usually referred to the region of the nipple on the corresponding side.

There are exceptions to this rule, however, which are sufficiently frequent and striking to merit particular attention. Thus the pain may be located in the back below the scapula, when it may simulate lumbago or the pain of acute nephritis. Various writers, notably Huss, have called attention to the occurrence of this symptom on the side of the thorax opposite to that showing the pneumonia, when it is probably due to the anastomosis of the right and left intercostal nerves. The anomaly in the distribution of the pain which is most singular and most liable to occasion error of diagnosis is the location of pain in the abdomen.

The suffering may be experienced in the epigastrium, the hypochondrium, the region round about the umbilicus, in the hypogastrium or either iliac fossa. When felt in the right iliac fossa the pain may be, and in fact has been, mistaken for appendicitis; and cases are on record, especially in children, in which laparotomy has been performed for a supposed appendicitis when the disease was subsequently found to be a pneumonia. Various explanations have been offered to account for abdominal pain in thoracic disorders (pleurisy and pneumonia), e. g., inflammation of the phrenic nerve which, according to Van Gehuchten, gives off sensory filaments to the costal and diaphragmatic pleura as well as to the pericardium and that portion of the peritoneum covering the anterior surface of the diaphragm.

Roher has suggested that inflammation of the diaphragm itself may be a cause of abdominal pain in pleuritis and pneumonia, especially in children. But the most plausible explanation is to be found in the anatomic fact that the lower six intercostal nerves supply the abdominal parietes as well as the lower part of the pleura lining the chest-wall and covering the diaphragm. Irritation of one of these nerves, as by pressure or inflammation, would cause pain to be referred to the abdominal wall, as in the case of the eleventh nerve to the iliac fossa.

I recently observed a pneumonic patient whose initial and, for a time, sole complaint was of intense pain in the abdomen below the umbilicus, though a day or two later the pain crept up into the right lumbar region. I recall also a boy of fifteen whose initial symptoms were epigastric pain and vomiting which strongly suggested gastric disorder, particularly as chill was either absent or so slight as to have escaped the patient's notice, while signs of lung involvement did not declare themselves for eighteen hours thereafter.

The pain of pneumonia is intensified by deep inspiration and the act of coughing, so that every possible attempt is made to suppress cough. There may be associated tenderness or cutaneous hyperæsthesia which makes examination of the lung distressing. The pain may also be aggravated by the act of turning from one side to the other for the purpose of physical examination, and I have known it to render prolonged lateral decubitus actually impossible, the suffering occasioned thereby serving to accelerate the pulse and respirations in a way to make one very uneasy.

The cause of the pain in croupous pneumonia is probably a concomitant pleuritis since it is wanting in centric pneumonia and tends to abatement as the inflamed pleural surfaces become separated by the exudate in the pleural cavity. For this reason the suffering generally disappears or becomes quite tolerable by the third or fourth day of the pneumonia, and yet it has been known to persist until the eighth or ninth day.

The early onset and intensity of this symptom add greatly to the patient's discomfort and often occasion considerable anxiety or even alarm on the part of both friends and patient. In particularly sensitive persons the persistence of severe pain may materially influence prognosis. In the senile form of fibrinous pneumonia pain may be, and often is, very insignificant.

Fever.—If the patient's temperature is taken during the initial chill it is found to be already elevated. It rises steadily and may reach its maximum within a few hours, even to 105° F. inside of nine hours after the invasion (Thomas). As a general rule the pyrexia may be expected to attain its greatest height from the second to the fourth day (fourth to sixth day, Fraenkel), although the maximum may be reached on the first or second or may very exceptionally be postponed as late as the tenth or even the nineteenth day (Wilson Fox).

In other cases the temperature rises gradually for the first three or four days and, according to the author last mentioned, may increase with a progressive advance in the signs of consolidation or may continue to rise after the disease has become stationary. Very exceptionally the fever may persist for only a few hours, and hence give rise to the erroneous opinion that pneumonia can exist without fever even when the lung changes continue. These are not typical cases and do not belong, therefore, to the sthenic form now being described.

Acute pneumonia displays a marked tendency to high temperatures and hence the fever generally ranges between 103° F. and 104° F. or even a degree higher, but may exceptionally go very much above this point. Of 503 cases of pneumonia analyzed by Wilson Fox with a view to determining the frequency of certain temperatures observed in this affection there was hyperpyrexia above 105° F. in 118, while it actually ran from 107° to 110° in five cases. In the greatest number of cases, 202, the fever-range was between 104° F. and 105° F.

The character of the pyrexia is not constant for all cases. In a minority of cases it may be continuous (Fraenkel), that is, it may show insignificant remissions of one half or perchance one degree C. (0.9° to 1.8° F.). In most instances the temperature-curve displays fluctuations of 1° C. to 2° C., or 1.8° to 3.6° F., and hence constitutes a distinctly remittent type of fever. The figures just given are from Fraenkel and, having the weight of so great an authority, are probably correct; yet I am inclined to regard such extensive fluctuations as more pronounced than are usually seen in the remitting form.

Occasionally cases are observed in which distinct intermissions occur and such intermissions may show no regularity, or the pyrexia may be as typically quotidian, tertian or quartan as in malaria (Preble). Such intermissions may or may not be accounted for by changes in the local signs, that is, by a commencing resolution corresponding to the fall in temperature and by fresh involvement of new areas (extensions) coinciding with the exacerbations of the temperature. Such vagaries in the fever-curve are often puzzling and apt to render the physician very uneasy lest they mean the formation of pus or complications.

The duration of the fever is also subject to considerable variation, but in the majority of cases it lasts from five to nine days from the date of invasion. In a considerable number of cases, according to Fraenkel, there occurs a marked decline of temperature on the third day of the illness, the fall occasionally reaching even to normal. Most commonly, however, the temperature shows a tendency to decline only after the fifth day, when it may sometimes be so pronounced as to constitute what is termed a pseudocrisis. The temperature then rises again for a day or two when it again falls more or less rapidly and steadily by crisis or lysis. (See Fig. 22.)

It should not be forgotten, however, that such a return of pyrexia after what appears to be a crisis may be owing to an extension of the disease. It is possible for several such deceptive intermissions and exacerbations to be caused by repeated extensions, as already stated.

Cough.—This symptom is probably never absent in sthenic cases and is generally one of the earliest features of the disease. It is not paroxysmal as a rule but short and dry, and, because of the intensification of the pain it occasions, is suppressed so far as the patient is able. Its frequency is variable, but for the most part it plagues the sufferer at short intervals. Indeed this symptom may be so pronounced and occasion so much distress as to necessitate special measures for its relief.

Expectoration.—This is usually quite characteristic in the sthenic form of pneumonia and by those unskilled in physical examination is much relied on as a means of diagnosis. It is rarely profuse, generally small in amount and sometimes so scanty as to occur only a few times in the course of the disease.

Its color depends upon the admixture of blood in greater or smaller proportion and is therefore somewhat variable. The sputum may be only streaked or speckled with blood, but in the majority of cases it presents the characteristic appearance known as *rusty*. In some instances the reddish hue is so faint that the color may be said to be orange or saffron.

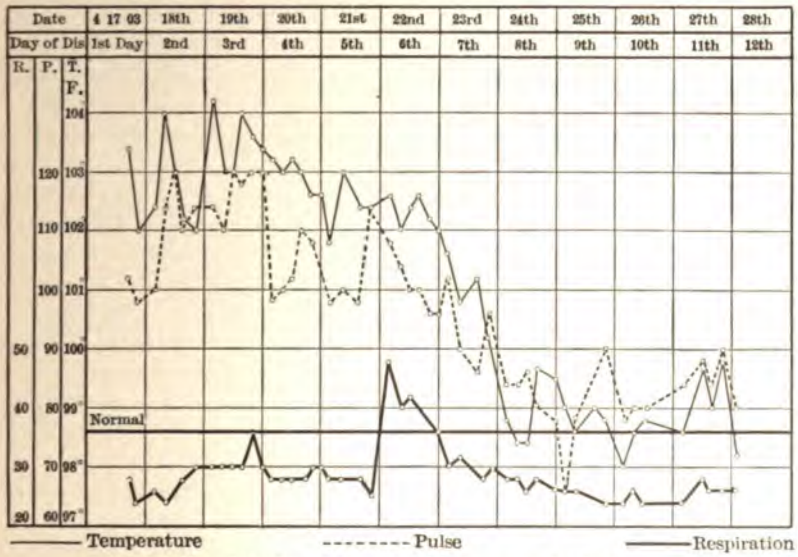


FIG. 22.—Chart of case with recovery by lysis.

The striking and distinguishing peculiarity however is not its hue but its extreme viscidity which causes it to be expelled with difficulty and to stick to the side of the sputum cup. Indeed such is its stickiness that the expectoration may have to be wiped from the lips or even taken from the
n v the patient's fingers, and the glass vessel containing it may be
side down without spilling its contents. The remarkable viscidity
is due to their richness in mucin. This tenacious quality of the
n is the feature that is to be specially noted, since although
r blood-tinged hue may fail, its glutinous nature does not.

this characteristic sputum appears in the early stage and lasts
n sets in, but it may be delayed until several days have passed

or, very exceptionally, as late as the tenth or even twelfth day (Wilson Fox). Very rarely it may first make its appearance immediately prior to crisis and then persist during the stage of gray hepatization and until resolution is completed.

If the pneumonia is attended by general bronchitis the rusty-colored expectoration may be obscured by that of the bronchial inflammation. In cases of great severity, particularly if they assume the asthenic type, the sputum may be more fluid in consistency and have a reddish-brown color like the juice of prunes. It is then known as *prune-juice* expectoration and is regarded with anxiety as indicating a grave form of the disease.

Microscopically the sputum is seen to contain epithelial cells of various kinds, mucoid cells, red blood corpuscles, leucocytes and, when properly stained, Fraenkel's diplococci. (See Plate I.) Another feature of pneumonic sputum worthy of note is its excess of chlorides in consequence of these salts being present in large amount in the pneumonic exudate. Their appearance in the expectoration explains their diminution in the urine, and conversely their reappearance in the urine during the stage of resolution accounts for their diminution in the sputa. Occasionally, as first shown by Remak, the expectoration may also contain minute fibrinous casts of the finest bronchioles and alveoli. With the setting in of gray hepatization, the sputa become more liquid, less sticky and rusty and finally disappear altogether.

Respiration.—Acceleration of breathing is always present at some time during the course of genuine fibrinous pneumonia but not to the same degree in all cases. During the stage of engorgement the respirations may not be hastened unless the intensity of the pain cause them to be somewhat more hurried as well as shallower. When, however, the exudate has formed, the increase in the rate of respirations becomes a notable feature and so out of proportion to the pulse-rate as to be of diagnostic value. Thus, with a pulse of 108 or even 120 the respirations may number thirty-two, thirty-six or forty. Since they normally bear the relation to the pulse of one to four, such differences as are cited above are spoken of as a *loss or perversion of the pulse-respiration rate.*

In primary and sthenic cases this disproportionate acceleration of the breathing is more pronounced than in the aged or in asthenic cases. Thus, with a pulse of ninety-six, the respirations may number twenty-eight, in itself not a conspicuous increase; yet in arriving at a diagnosis in obscure cases such an augmentation may be of very great significance. It must be borne in mind also, as already stated, that this loss of pulse-respiration ratio may be slow in appearing and then seems to depend upon tardiness in the formation of the exudate, since it first becomes apparent with the recognition of dullness and distinct bronchial breathing. Moreover, the degree of frequency of the respirations seems to be determined by the extent of lung involvement. Such cannot be the only factor in its production, however, since in any given case the respiration-rate may evince considerable fluctuation from time to time and even from hour to hour.

The subjective sense of dyspnœa varies in different cases, but, for the most part, patients have an appreciable difficulty in obtaining sufficient air.

It is not a good sign when a patient is unconscious of his dyspnœa since it betokens more or less blunting of his sensibility. If, as sometimes occurs, there is intense bronchitis with accumulation of secretions in the air-tubes, and the patient is yet unconscious of such accumulation, it indicates from a prognostic standpoint a grave loss of reflex excitability. As death approaches the degree of carbon-dioxide poisoning is such that although breathing may be difficult and the chest may emit a multitude of râles the patient appears actually benumbed. I have seen such a case fail to respond to a large hypodermic dose of apomorphine given in the forlorn hope that emesis might clear out the tubes.

To return to the subject of the loss of pulse-respiration rate in croupous pneumonia, one should carefully distinguish between increased frequency of breathing seen in cardiac or chronic pulmonary disease or in extensive pleuritic effusion and that seen in the affection we are now considering. In acute pneumonia the pulse is retarded out of proportion to the height of fever and extent of hepatization, whereas in other affections causing dyspnœa the heart's action is likely to be accelerated. Hence the perversion of pulse-respiration ratio seen in pneumonia depends also on the disproportionate slowing of the pulse.

Whereas the respirations in most instances run from thirty-two to forty, a far greater increase than this may be recorded. In children particularly the breathing may be very rapid, sixty or even higher. In adults, also, some temporary disturbing factor may markedly accelerate respirations, to forty-eight, fifty or perchance sixty, but such a rate is rarely maintained for long. In my experience such disturbing causes have usually been a fresh accession of pain due to extension or involvement of the opposite lung.

Finally, one should bear in mind the possibility of extreme rapidity of breathing in hysteria. I once witnessed a striking instance of the kind out of all relation to the pulse. Such cases cannot be mistaken for acute pneumonia if due attention is paid to history and associated symptoms.

Circulatory System.—The pulse-rate is always more or less accelerated in fibrinous pneumonia depending on conditions residing in the heart itself or on the effect of the poison on the myocardium. In the beginning the pulse is full and bounding and of increased tension, but with progress of the disease it loses in force and volume even when not materially increased in frequency. Dr. E. F. Wells believes that arterial pressure is low from the start and regards this lowness of pressure as an attempt on the part of the system to combat the infection by causing a pouring out of leucocytes.

As the pneumonia advances the tension of the pulse grows less as determined not only by the sphygmomanometer but by the palpating finger. This increasing loss of tension is a feature on which Fraenkel out of his great experience lays much weight. It is due not alone to failure of heart-power but depends in large measure on decrease in vascular tone. As I have often observed capillary dilatation may be shown by pulsation in the finger-tips when these are grasped. As vigorous systoles are requisite to the production of capillary pulse, it will be observed that with failing strength on the part of the heart this throbbing of the fingers disappears.

If the heart-muscle remains unimpaired and proves equal to the strain, the rate of the pulse remains not greatly increased and not as high as would naturally be expected from the degree of the pyrexia and from the rapidity of the respirations. In favorable cases the average pulse-rate is not far from 110 or 120; but should the malign influence of the infection be expended upon the myocardium, the rate may, and generally does, exceed the latter figure. According to Sear's and Larrabee's tables the mortality appears to stand in direct relation to the frequency of the pulse, a pulse-rate of 130 or higher furnishing a very unfavorable prognosis. It should always arouse apprehension, therefore, whenever the pulse is unduly frequent from the outset or tends to become so with progress of the disease.

I recently saw in consultation a fatal pneumonia in a man of fifty whose pulse was more than 120 from the beginning and on the fifth day was 150. It was the one feature of the case that was ominous and although the pulmonic second sound was still strong, the prognosis was considered most grave, and in fact death occurred in less than thirty-six hours thereafter. Irregularity or intermittence of the pulse is likewise of serious import. I have in mind a man of about fifty whose pulse suddenly became arrhythmic during the examination apparently from the strain of sitting up for a few moments that the posterior bases might be compared. Although he was laid down immediately his pulse did not again grow regular, and death took place a few days subsequently.

The volume and strength of the pulse are also determined in a measure by the extent of lung involved. If the exudate is extensive, large areas of pulmonary vessels are practically cut out of the circulation and the supply of blood to the left heart is correspondingly diminished. The pulse must, accordingly, become small and weak and the right heart overdistended.

Cyanosis is another feature connected with the circulatory system which may show all degrees of severity from slight blueness about the nails and lips to a general duskiness, particularly of the countenance. This symptom is still attributed by most physicians to defective hæmatosis and is thought to indicate a resort to oxygen-inhalations. As a matter of fact cyanosis is a manifestation of capillary paresis depending, as shown by Romberg and Paesler, upon toxic paresis of the vaso-motor centers in the spinal cord. This condition sometimes develops early in the disease, by the third or fourth day, and although in its milder degrees is not incompatible with recovery, it should when it increases be regarded as a danger-signal if indeed not an omen of impending disaster. To the experienced practitioner pronounced cyanosis in acute pneumonia is a matter for grave apprehension. Its appearance should be carefully looked for in all cases so that, upon its earliest recognition, prompt and energetic measures to sustain the heart may be instituted.

Blood.—Much study has been given to the blood in fibrinous pneumonia, with a view to determining the presence of micro-organisms in the circulating fluid and to ascertaining what changes, if any, take place in its corpuscular elements. Cultures from the blood were at first very inconstant and unsatisfactory. Thus, the Klemperer brothers never found pneumococci in the blood, and Fraenkel also failed in 80 per cent of the cases so long as he used the agar-plate method, but succeeded in all cases by using bouillon as the culture medium. Another reason for the failure of early investigators lay in the abstraction of too small quantities of blood (5 c.c.). When, despite faulty technique, the diplococcus was obtained it was in very severe, usually fatal cases (White, Cole), and the conclusion was drawn that the presence of pneumococci in the blood should be regarded as of evil prognosis.

It may now be stated that with improved methods the diplococcus may be detected by cultures and even in smears in the blood of practically every case of acute lobar pneumonia. Rosenow's investigations along this line were so complete and his contribution to this subject is so valuable that I cannot do better than quote from his paper which appeared in the *Journal for Infectious Diseases,* March 19, 1904. This investigator, whose work was carried on in the laboratory of Rush Medical College, after carefully disinfecting the skin of the arm took 7 c.c. of blood in early cases and 10 to 13 c.c. in later ones. "With great care to avoid contamination, so far as that is possible, 5–7 c.c. of blood were used for cultural purposes, while the rest was transferred to a 50 c.c. graduate, slanted until the blood coagulated, and then put into the ice-chest upright to permit the clear serum to drain off, which was then used for agglutination and other tests."

Of 145 cases positive results were obtained in 132. In two cases (Nos. 35 and 24) the cultures remained sterile, but smear preparations directly from the blood showed the presence of undoubted pneumococci. "Otherwise positive results were obtained in all stages of the disease; in one instance, where pneumonia developed in a patient in the ward, the organism was obtained in pure culture twelve hours after the initial chill, before there were any definite physical findings." "The pneumococcus was demonstrated in the blood in four other cases before a positive diagnosis could be made." "The fourth, fifth, and sixth days gave the highest percentage of positive results, except the period within the last twenty-four hours before death in the fatal cases."

At present writing Rosenow's series has been increased by thirty additional cases, making 175 in all, with positive results in 160, or over 91 per cent.

Briefly stated Rosenow's conclusions are as follows: With improved methods of culture and sufficiently large quantities of blood the diplococcus may be demonstrated in all cases of croupous pneumonia. The presence of this organism does not necessarily indicate an unfavorable prognosis. Examination of the blood by cultures and smears is a valuable means of diagnosis in cases of pneumococcæmia which do not furnish signs of lung involvement. There appears to be a diminution in the number and viability of the pneumococci in the blood at the time of crisis, but they may be obtained for as long as forty-eight hours after crisis. Rosenow has also investigated the agglutinating power of the blood and found clumping to take place with a dilution of one to fifty, yet, owing to difficulties in the way of this test, he regards it as of scientific rather than practical value.

Other of his observations are of much interest and have a possible bearing upon treatment. He found that the voluminous precipitate produced by the growth of the pneumococcus in serum of pneumonia patients is owing to the presence of acids. Accordingly, Rosenow queries if the pneumococcus by its growth in the consolidated lung and in the patient's blood during life does not produce acids the same as in the test-tube; and if so, may not some of the symptoms of pneumonia be due to an acid intoxication? Such a view seems corroborated by the reports of other investigators that late in the disease, when the power of resistance is reduced, after crisis and after death the blood has been found of diminished alkalinity. This will be referred to again in considering treatment.

The *coagulability* of the blood of pneumonic sufferers is increased, the clot forming very rapidly and being remarkably thick and firm. It is for this reason that heart-clots are so generally found after death and that sudden death, occurring when the patient appears doing well, is attributed by some clinicians to the formation of an *ante-mortem* clot in the cardiac cavities.

The *specific gravity* of the blood is said to be high throughout the course of the disease, particularly in children. In other cases it is reported high only when there is cyanosis and the blood is obtained from peripheral capillaries. Under these circumstances the corpuscular elements are said also to be increased. The *toxicity* of the blood is also said to be doubled (Cabot).

The *red cells* are said to show changes in both their number and appearance, although the latter is not constant. It is certain, however, that the erythrocytes diminish in number corresponding to the height of the pyrexia so that in cases with high fever anæmia becomes rapid and pronounced (Rosenow). Others attribute this anæmia to the precipitation of the red cells out of the blood into the pulmonary exudate, to which fact is attributed also the diminution of the chlorides in the urine.

The *white cells*, on the other hand, are generally increased, often to a remarkable number, twenty, forty, fifty thousand or more. The average may be put down at from twenty to thirty thousand. This hyperleucocytosis is due to increase of the polymorphonuclear elements chiefly, which may constitute 90 per cent of the entire number. Eosinophiles and blood-plates are wanting, while the lymphocytes are reduced. After crisis, however, the polymorphonuclear cells decrease and may fall to below 50 per cent, while eosinophiles and blood-plates become more numerous than normal.

This leucocytosis is a manifestation of an attempt on the part of the organism to resist the infection, and therefore it would seem as if a reliable indication as to the outcome of the malady ought to be furnished by the degree of the leucocytosis. However, this is not the case. Both an intense infection and a vigorous reaction are necessary to the production of a marked leucocytosis, for, according to Cabot, there may be no increase of leucocytes in a mild infection with strong reaction the same as in a violent infection with feeble reaction. If leucocytosis is to be pronounced there must be both vigorous resistance on the part of the animal organism and an infection of considerable severity. Even in such a case the issue must remain a matter of doubt until the struggle is really over, for it is an open fight between the organism of disease on the one hand and the ability of the patient to resist on the other.

A fact of utmost importance and one to be remembered when considering treatment is that we possess no means of increasing the leucocytosis, and that the administration of coal-tar antipyretics diminishes the number of the white cells (Hare). Moreover, we are not yet certain which of the forms are specially lacking in cases that succumb, since the published data are, according to Cabot, quite conflicting. The bearing of leucocytosis on prognosis will be alluded to again in the proper place.

Rosenow concluded that the degree of leucocytosis in pneumococcus infection may be regarded as an index of the degree of resistance, and that the leucocytes probably constitute an important factor in combating the infection. He thinks also that a high leucocytosis furnishes a favorable prognosis and that when hypoleucocytosis succeeds a primary increase it is of bad import as indicating an exhaustion of the resisting power of the individual. It may be added that Dr. M. W. Hall has investigated the blood of pneumonia patients in the light of Arneth's studies of the blood in acute infections. (See *Blood* in Pulmonary Tuberculosis.) Hall found the polynuclear neutrophiles tended to disappear, being replaced by the cells showing one, two or three nuclei. In one fatal case the neutrophiles consisted almost exclusively of those belonging to the first and second groups.

Skin.—This usually feels pungently hot even when moist. It may be dry but in most cases is more or less perspiring. As regards sweating during the pyrexial period, cases differ much. Perspiration may be present throughout the course of the disease or it may be absent during the height of the pneumonia, or it may occur only at the time of the crisis. In the latter event it is generally profuse and is a warm sweat. Although a warm perspiration is the rule in sthenic cases, and by Andral and others was regarded as of favorable augury, still, pronounced moisture of the skin is more common in asthenic cases, especially in alcoholics. A cold sweat with evidence of depression late in the course and before crisis is a bad omen since it indicates collapse and calls for energetic stimulating measures.

Herpes occurs so frequently in croupous pneumonia (13 to 43 per cent, Juergensen) that, in a febrile state, its presence justifies a suspicion of pneumonia (Wilson Fox). Its most usual seat is about the lips but it may appear on other parts of the body as the arms (Thomas), the nose, genitalia and anus (Osler), or over the whole trunk as observed by Peacock in one case. This eruption does not usually appear before the third or fourth day and in some instances is postponed until the crisis or during convalescence.

Other eruptions may be observed in exceptional cases, as *acne* noted by Traube and *urticaria* seen by both him and Schroetter. Fismer reported *boils* in five out of 230 cases, while Juergensen is stated by Fox to have witnessed "*multiple cutaneous gangrene.*" *Capillary hæmorrhages* into the skin and *purpuric* spots have also been observed in cases of great severity, while *sudamina* or a *miliary rash* that may even be followed by desquamation may occur when perspiration is profuse.

During the Winter of 1904 I observed a male patient in Cook County

Hospital who, among several other unusual manifestations, developed on the tenth day of his illness four circumscribed abscesses which closely resembled furuncles.

One of these boils, for such they might be called, was situated on the anterior surface of the left upper arm not far from the elbow, a second considerably larger on the right forearm about midway between elbow and wrist, and the third on the anterior aspect of the left leg below the knee and the fourth on the left thigh. They looked so like abscesses from hypodermic injections that the nurse was at first held responsible for their production. Inquiry of the internes and nurses brought out the fact, however, that no injections had been given in these situations. Unfortunately these abscesses broke and discharged their contents before the interne had gotten ready to make cultures from their pus. When my attention was called to them they were scabbed over and presented the appearance of ordinary boils that had ruptured.

This patient is also reported to have had, at an earlier period in his disease, a disseminated eruption on the back which consisted of minute red papules that looked like sudamina. This case will be found more fully narrated at another place.

The bright flush previously mentioned as seen on the cheeks and often most marked or even present only on the side corresponding to the lung involved, is thought to be specially associated with this disease, and I have known practitioners to attach much diagnostic importance to its unilateral appearance. Its presence possesses a certain value but its absence does not, on the other hand, weigh against the pneumonia. I have recently seen a case of acute tuberculous pneumonia of the left apex in which there was a bright red spot on the left cheek. It may be regarded, therefore, as a minor symptom of pneumonic inflammation but not by any means of croupous pneumonia.

Digestive System.—Vomiting is an early symptom in some cases, particularly in children, and when observed may mislead the unwary into the diagnosis of a gastric disorder. Usually there is nothing more than anorexia. The tongue is generally dry and covered with a white coat, while in severe cases the lips may be parched, fissured and coated with sordes. The dryness of the mouth is intensified by the fact of the patient's breathing through the mouth on account of his dyspnœa. The breath is offensive and has often seemed to me to possess a peculiar somewhat aromatic odor.

Thirst is more or less intense, and yet a very sick pneumonia patient may not call for fluids, in part, perhaps, because the sensibilities are obtunded by the toxæmia. The bowels are usually confined and are very apt to be distended with flatus. Diarrhœa is sometimes present and is one of the features characterizing the form known as bilious pneumonia.

A symptom of such grave influence as to be justly dreaded is septic diarrhœa in which the dejections are frequent and of a foul odor. It is usually accompanied by meteorism and is most difficult of control.

Such a diarrhœa may set in early but does not usually appear until after several days. It is to be regarded as something more than a putrefactive decomposition of the ingesta, being probably a manifestation of the intensity and virulence of the infection which has attacked the gastro-intestinal tract. I have seen such a state of things in association with other evidences of profound toxæmia and with such a degree of tympanites as suggested paralysis of the bowel. It has generally been my observation, however, that when paralytic distention does occur it is difficult to get the bowels to act in response to even strong cathartic remedies.

The liver is more or less engorged and may be palpable. This is the case in young children, but even in adults the organ may be tender. The spleen is also enlarged and may be palpated when the abdomen is not too distended and firm. There is nothing in this fact peculiar to croupous pneumonia, however, since the organ is palpable in most acute infections of severity.

Nervous System.—As remarked by Aufrecht it is the nervous system which, next to the heart, plays a prominent part in the symptomatology of acute pneumonia. In most cases there is no more disturbance than is shown by headache, restlessness and insomnia, but convulsions and delirium are not uncommon. *Headache* is experienced by the great majority of patients, having been absent in only eight of Louis' forty-seven cases, but in severity great differences are observed. As a rule it is not so intense as to furnish occasion for special complaint. In some it may be agonizing and may become wellnigh insupportable during paroxysms of coughing. The headache is usually frontal and is said to be an especially prominent feature of apex pneumonias. It is apt to be an early symptom and may even usher in the illness, as in the following case in which it was the initial manifestation.

December 30, 1904, I was called to Ottumwa, Iowa, to see R. S., a young man of sixteen, who was the victim of a congenital heart affection and had been seen by me on two previous occasions. From Dr. William Hansell, the attending physician, the following history was obtained. Some ten days earlier the young man had consulted Dr. Hansell on account of a troublesome pain located in the right side of the head. Excepting a conjunctivitis of the right eye nothing was discovered to account for the headache, and after that had been appropriately treated and relieved the pain had gradually disappeared.

Nothing further was heard of the patient until early in the morning of Tuesday, December 27th, when the doctor was summoned because of a return of the headache. At this time the pain was agonizing and, as before, located in the right half of the head. The temperature was 100.4° F. In spite of remedies the cephalalgia continued with great intensity and slight fever persisted. On Thursday, the 29th, the temperature rose to about 103° F. and on the day immediately preceding my arrival at midnight had mounted to 104° F. The respirations which had previously been twenty rose to forty while the pulse ranged in the neighborhood of 100.

During the two previous days there had been noted a tendency to mild delirium, although the patient recognized those about him and answered questions rationally. I found him lying on his back with his head turned to the right and buried in the pillow. His suffering was so intense that he moved his head only with great reluctance. The temperature, pulse, and respirations were as stated above. Cough was so insignificant as to have attracted no attention. He had had no chill and in the way of pain other than the headache, had complained a day or two before only of a slight ache in the left side of the back.

Upon examination the heart showed the anomalous signs which had previously led to the diagnosis of a congenital cardiac disease. There was nothing in this, however, or in the head symptoms to account for his fever or his accelerated respirations, and as there was a marked loss of normal pulse-respiration ratio the lungs were suspected to be the real seat of the mischief. Accordingly, on going over the chest, I discovered dullness with bronchial breathing in the left infrascapular region along its outer aspect. There were no crepitant râles, but the signs were considered sufficient for a diagnosis of acute croupous pneumonia of the left lower lobe, which had required three days to come to the surface sufficiently for recognition.

The patient died in the early morning of January 1st, the fifth day of his illness. The *post mortem* disclosed red hepatization of the left lower lobe and a malformation of the heart, the aorta taking its origin from both ventricles with hypoplasia of the pulmonary artery and two rudimentary tricuspid leaflets.

Insomnia is very frequent in fibrinous pneumonia and when pronounced and obstinate exerts a malign influence by robbing the system of needed rest. In cases in which the nervous system or cerebral centers are less profoundly affected by the toxæmia the sufferers doze off into short unsound naps from which they are easily aroused by cough or other disturbing influences. The opposite condition, i. e., pronounced sleepiness, is said by Aufrecht to be a very serious symptom since the patients who manifest such somnolence rarely recover. It should be stated, however, that this author has observed it in children when it did not possess such evil import.

Convulsions are rarely observed in adults, and yet a man recently admitted to Ward 4, Cook County Hospital, with pneumonia of right lower lobe, displayed repeated epileptiform seizures which at first disguised the real nature of his malady. An autopsy could not be obtained. In children this symptom may initiate the illness and does not then possess especial significance since the nervous system of the young child is most sensitive to disturbing factors and especially to acute infections. It is quite another thing, however, when convulsions set in late in the disease, since they probably indicate involvement of the meninges and generally prove fatal (Holt). I recently saw, in consultation with Dr. Frank S. Churchill of Chicago an infant of thirteen months who, on the tenth day of her pneumonia, when the temperature had fallen somewhat and the doctor looked upon the general condition as promising recovery, suddenly, at one o'clock at night, had a severe general convulsion. Appropriate measures were at once instituted, but without avail, as two more spasms occurred in rapid succession, death resulting in the third.

Delirium is a very common feature of acute pneumonia and merits particular attention as regards its severity, its significance, and its time of occurrence. It may show all grades of intensity from a low muttering form to one of such severity as to resemble an acute mania and to necessitate restraint of the patient, or unremitting vigilance on the part of the nurse lest the patient cast himself from a window, as has happened more than once. Impulse to such an act may result from the delusion that the invalid is being persecuted, and Aufrecht gives an account of a man, who up to that time perfectly quiet, suddenly sprang out of bed, cast the bedclothes to one side, and seized a small table with which he burst open an adjacent window. He then threw the table out of the window and before the attendant standing but a few feet distant could divine his intention sprang out after it into the court below. The entire scene was witnessed by other patients and yet so rapid and unexpected were the man's movements that they could not have interfered had they suspected his design.

Low muttering delirium is most frequent in pneumonia patients who have been addicted to alcohol (10 to 12 per cent, Huss). It closely resembles the delirium of *mania a potu*, being its initial stage, as it were (Aufrecht). This form of mental wandering is very common in asthenic cases even where there is no chronic alcoholism, and is frequently seen in the aged. It is usually considered a bad indication and calls for stimulation.

Another form of delirium is that which occurs during the acme of the pneumonia or even at its beginning. As it is most often seen in sthenic cases with high fever, it was attributed, especially by older writers, to the elevation of temperature *per se*. Most clinicians now regard it as due to the action of toxines on the cerebral centers and not to the fever, since it may persist during remissions in temperature and is sometimes present in cases not characterized by intense pyrexia.

On the other hand, delirium may be absent in pneumonia patients who show more than the usual degree of fever, as observed by me this past Winter in a man who, notwithstanding an average temperature of 105° to 106° F., remained perfectly free from even a semblance of delirium. As with headache it appears to be more common in pneumonias of the upper than the lower lobes, and hence Fraenkel suggests that its cause lies in derangement of circulation within the brain. He thinks that consolidation of the upper lobe may by pressure on the great venous trunks (superior vena cava and innominate) interfere with the return flow of blood from the interior of the cranium, an explanation which is ingenious to say the least.

Still a third form of delirium is that due to *exhaustion*. This is a late symptom and may appear immediately prior to crisis, during crisis, or after defervescence has become established. It is the fact of its occasional occurrence after crisis which supports the assumption that its cause is exhaustion and not toxic. Furthermore, it yields to stimulation even when appearing just before crisis which the delirium of toxæmia does not.

To recapitulate, three types of delirium may be encountered in acute pneumonia: (1) the low muttering form, seen in drunkards chiefly but also in the asthenic; (2) the toxic or febrile form, occurring usually during the acme of the disease when pyrexia is high, but likewise during remissions in the temperature, and often absent in cases characterized by intense fever; (3) the delirium of exhaustion or inanition (Fraenkel) which is a late manifestation and is apt to be very active, even maniacal. All types are serious and affect prognosis unfavorably.

Strictly motor disturbances are few or wanting. Restlessness is not uncommon and may be united either to slight benumbing of the faculties or to unnatural clearness and alertness of the sensorium. Aufrecht cites Stephan and Lepine as having noted *hemiplegia* in a few cases, no satisfactory explanation for its occurrence being adduced. That the paralysis appears to be toxic or of reflex vagus origin and not due to pressure on the brain substance, seems borne out by the failure to find definite anatomical lesions in the cases that have been investigated *post mortem*. The hemiplegia appears to have been observed most frequently, although not exclusively, in old persons, and hence local ischæmia of the brain has been suggested as a possible factor in conjunction with some of the other influences mentioned. Lastly, rigidity of the neck has been recorded in young children, even without meningitis, which latter, when it occurs, is to be regarded as a complication and not a symptom of a disordered nervous system properly speaking.

Urine.—As in other febrile affections the urine is diminished in amount and increased in specific gravity to 1.030 or 1.035, this increase being due partly to lessened excretion of water and partly to excessive elimination of urea and uric acid. Osler states that these substances are increased at first, lessened during the height of the disease and again augmented after the crisis.

The sulphates are increased and the phosphoric acid is diminished and urates are deposited in excess. Albumin is another not infrequent ingredient of the urine and is due to acute glomerular nephritis consequent upon the passage through the kidneys of toxins and pneumococci. The albuminuria is usually slight, and may be associated with the presence of epithelial casts and even blood. This nephritis may, according to Wilson Fox, be a late phenomenon and occur now and then during convalescence.

This may be a grave complication in the old, particularly in those who have chronic nephritis, since it may lead to a serious retention of urea and other solids, and interferes with the elimination of the toxins of the affection. On the whole, however, acute nephritis is not so frequent or severe in pneumonia as in infections caused by streptococci.

Diminution of the chlorides is the most striking characteristic of the urine of acute croupous pneumonia. It is owing to the retention of the chlorides in the pneumonic exudate, a fact shown by their increase after resolution and their decrease or actual disappearance again upon the occurrence of an extension. For this reason daily estimation of the chlorides is of aid in detecting resolution or repeated extensions.

This explanation of the diminution of the chlorides is proven by a case of pneumococcæmia which is under observation at the present writing. The lungs are entirely free from involvement and the estimation of the chlorides by Purdy's method showed 18 gms. in twenty-four hours, a full amount.

Blood-cultures in this case resulted in the growth of pneumococci in pure forms. The organisms were agglutinated by the blood of the patient, thus showing the germs to have come from the blood.

Crisis.—The striking suddenness with which lobar pneumonia sets in is paralleled only by the abruptness of its decline. This sudden abatement of what, until then, was a violently raging disease is known as the crisis. It is characterized by a rapid fall of temperature to normal or even below, this complete defervescence taking place within twelve and sometimes a few hours. Coincidently with the drop in temperature the patient breaks out into a profuse perspiration, his pulse and rapid breathing grow slower and he falls into a refreshing sleep, to awake a few hours later at ease and ready to take nourishment. In most cases this crisis is not preceded by any premonition of its occurrence, while in others there is a precritical fall of a few degrees in the fever, after which the temperature again rises, to subside permanently on the following day. In other cases the crisis is succeeded by a brief and unimportant rise of the temperature.

The day of the illness on which crisis is likely to take place has been a subject of much discussion and investigation. By early writers and even by Traube it was thought to occur, as taught by Hippocrates, on the uneven days, the third, fifth, seventh, ninth etc. But, as emphatically stated by Liebermeister, this is now known to be incorrect.

Crisis may appear on any day from the second to the eighteenth or even later, but in the majority of cases from the fifth to the ninth. Of 721 cases tabulated by Juergensen the crisis took place on the uneven days in 442 and on the even days in 279 cases, while it fell on the seventh in 165 cases or 22.9 per cent and on the ninth day in seventy-two cases or 10 per cent, on the sixth day in eighty-seven cases or 12.1 per cent and on the eighth day in ninety-one cases or 12.6 per cent. His figures show, therefore, that of the whole number, 535 cases or 64.2 per cent experienced the crisis from the fifth to the ninth days inclusive. Of 366 cases gathered by the Collective Investigation Committee and given by Wilson Fox in which the crisis had occurred by the eighteenth day, this fell on the even days in 167 cases, leaving 199 in which it took place on the odd days. It is also worthy of note that of this number the crisis occurred between the fifth and ninth days inclusive in 277 cases. These tables certainly bear out the contention that the crisis is more likely to occur on the odd than on the even days, but what is still more important they show without doubt that in the majority of cases it falls between the fifth and ninth days. Dr. Edward F. Wells is of the opinion that the belief that the crisis occurs more often on the odd than on the even days is an error, based on inaccurate observations, and yet the following figures taken from his elaborate collection of statistics which were tabulated to show the duration of the disease, the cases having probably terminated by crisis, would appear to controvert his opinion. Of his own cases, however, 244 came to a crisis on the even and 200 on the uneven days. Of the nineteen authors he quotes there were 4,201 cases, of which 2,614 terminated on the uneven days and 1,587 on the even days. Tabulated according to duration of disease (probable crisis), his cases are as follows:

1st D.	2d D.	3d D.	4th D.	5th D.	6th D.	7th D.	8th D.	9th D.	10th D.	11th D.	12th D.
10	38	178	201	573	499	843	497	435	242	205	370

After all, it is not so important to know whether the crisis is likely to fall on an even or an uneven day as it is that the disease generally rages about a week depending on conditions residing in each case. The frequency with which convalescence sets in between the fifth and ninth days is brought out by the figures just given in a striking manner, and hence the important fact to be borne in mind is that a turn for the better may be expected at any time after the beginning of the fifth day, but on what day it is going to occur no one can predict with certainty. If the illness runs longer than nine days it is rare for it to terminate by crisis (Wells) and this may be said to grow progressively less likely with each day succeeding the seventh.

Various conditions influence the probability of a critical ending to the case. If the exudate within the lungs takes place all at one time and does not spread at repeated times, then resolution is likely to begin in the whole mass at once and crisis will probably take place. If, on the other hand, new areas become involved as others previously affected begin to resolve, the course of the disease is protracted and may be marked by sudden declines of temperature that represent attempts at crisis which is prevented, however, by the fresh involvements.

A critical defervescence may also be prevented by complications such as empyema, to cite the most common example. At the end of about a week temperature drops, but rises again after a few hours, and thereafter drags on in a tedious fashion for several weeks ranging rather irregularly from 99.8° to 100.5° F. in the morning, to 101° or even 102° F. in the afternoon. Such cases should always be regarded with suspicion and necessitate repeated careful search for complications.

Probably also the power of resistance on the part of the individual has much to do with the occurrence of crisis. What constitutes this ability on the part of the organism to react we know only vaguely. The individual may have been weakened by previous disease, by age, natural delicacy of physique etc.; but certain it is that in such persons pneumonia is less likely to end by crisis or that this, when it occurs, is less pronounced than in the robust. There may likewise be such virulence in the disease itself or in the bacterium as to prevent the development of the antitoxins on which the occurrence of crisis depends.

Lysis is the term applied, therefore, to the slow gradual and not abrupt cessation of the pyrexia. Instead of signs of resolution appearing in the lung at or near the time of crisis they begin to disclose themselves by degrees, beginning at about the time the disease ordinarily ends and extending through a period of several days or a week. During this time the pyrexia declines slowly but steadily and the patient's general condition undergoes a correspondingly gradual improvement. It is generally thought that such a termination is less frequent than is that by crisis, but it is likely that gradual return of temperature to the normal is more common than the description of this disease would lead one to suppose. When, in a case complicated by purulent pleurisy the pyrexia drags on in a tedious manner for several weeks coming to an end by lysis, apparently, it will generally be found, according to my observation, that a careful study of the temperature chart reveals a sudden remission perhaps even intermission somewhere about the end of the first week which really marks the time when the resolution of the exudate within the lung began. It is generally noted, too, that at this time the patient's pulse and respiration show some change for the better; and although the presence of the pleuritic exudate obscures the detection of the resolution, the lung has in reality been clearing up, as evinced by the loss of the characteristic sputa. No doubt many more cases of pneumococcus empyema which underwent gradual absorption were formerly mistaken for instances of slow resolution of the pneumonic exudate than is now the case. Such errors are, however, likely to occur and one should be on his guard lest he confound such a case, with termination by lysis of the inflammatory process within the lung. It is in children particularly that an empyema is likely to be overlooked, and, on the other hand, one should remember that in them crisis is very likely to be delayed, which circumstance has more than once made me suspicious that the crisis had been masked by an empyema.

The following case presents so many of the features that have been dwelt on in discussing the symptoms of acute pneumonia, that it is thought best to narrate it at some length. C. S., an Italian laborer, aged thirty-eight years was admitted to Ward 10, Cook County Hospital, March 11, 1904. Previous illness including venereal disease was denied and his use of alcohol had been moderate. He had not felt like work for a month, but had been really ill for only five days, with loss of appetite, repeated vomiting and pain in the lower half of the abdomen. Cough had been slight and expectoration scanty. He had experienced a pronounced chill the night before admission, had had five bowel movements the morning of the eleventh and thought he had been feverish for the five days these prodromal symptoms had existed.

Examination showed a well-nourished man with flushed face, a small rapid regular pulse, no apparent dyspnœa, but temperature of 104° F., hot, dry skin and great difficulty in turning from one side to the other on account of pain in the abdomen. Over the right lower lobe were dullness, bronchial breath-sounds, increased vocal resonance and crepitant râles, the rest of the lungs and the heart being negative. The abdomen was tender to pressure over the right iliac fossa, rather tympanitic and free from eruption, while liver and spleen were not palpable.

The accompanying chart (see Fig. 23) shows the range of temperature for the next ten days. It will be observed that up to the evening of the 14th the pyrexia was high, fluctuating about 105° F., and on the afternoon of the 13th reached 106° and 106.4° F., while from the afternoon of the 14th on, it gradually declined until the temperature became normal on the 18th. It is worthy of note that, notwithstanding the height of the fever, the patient's intellect remained perfectly clear. The pulse-rate was rapid, running for the most part from 120 to 132 excepting on a few occasions when it dropped to 108 or thereabouts, usually after a sponging. The respirations ranged from twenty-eight at time of admission to forty on the 13th, and in this regard exhibited acceleration out of proportion to that of the pulse.

On the evening of the 19th his temperature again rose, reaching 103.8° F. on the evening of the 20th, and remained about the 103° F. point on the 21st. This return of pyrexia was found coincident with and therefore due to an extension of the pneumonic process as shown by dullness, bronchial breathing and crepitant râles in the extreme upper portion of the right lower lobe behind, immediately above the superior limit of the original area. On the 23d the temperature fell to 98.6° F. at 9 A.M. and from that date on until April 1st fluctuated between 99° and 100° F., becoming permanently normal about April 4th. Patient was discharged on the 12th, well.

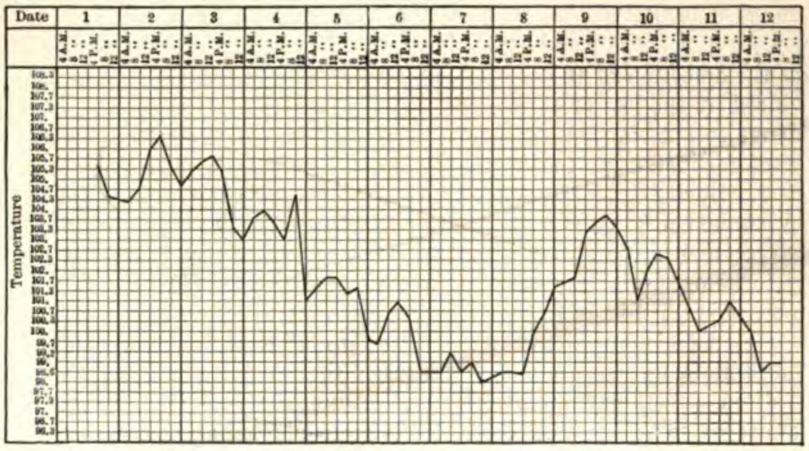


Fig. 23.—Chart of temperature in case of C. S. narrated on page 231.

Besides the extension of the pneumonic process there were several other features of unusual interest in this case. First of these, was the occurrence of abdominal pain which was so prominently complained of that had the chest not been examined it might have readily led to an error in diagnosis. When I saw the patient the morning after his admission he did not, at first glance, present the ordinary aspect of a pneumonic patient. He was not coughing, did not appear dyspnœic, although his respirations were accelerated, and with his high temperature and abdominal pain he strongly suggested a case of typhoid fever or appendicitis. Later in that same day, however, he began to complain of pain in the right side of the chest which at length grew so intense as to require the application of an ice bag. Thenceforth, although on several occasions, notably at the time of the extension, he spoke of pain in the abdomen, it was subordinate to that in his chest.

The second point of very unusual interest in this case was the development of boils, which have been described already in discussing symptoms referable to the skin. These abscesses were discovered and noted in the record on the 20th and might, therefore, be supposed to account for the rise in temperature recorded for the few days following the 19th. The occurrence at this same time of the extension of the pneumonia was very obvious, however, and probably accounted for the aggravation of symptoms, especially since it was ushered

in by pain in the abdomen and later on in the chest, precisely as at
ning of his illness. The subcutaneous abscesses may have served t
the temperature, although on this point we can only conjecture.
red eruption on the back, also previously mentioned in connection
nomena pertaining to the skin, was first observed on March 7th.

The third point of interest was the long continuance of a low irre
which dragged on for a week or ten days after resolution had set in.
this was owing it is difficult to say, since the lung was clearing up sat
but it serves to emphasize the statement already made that there m
fever, even in cases which display a fairly well-marked crisis. A dis
cal defervescence took place and marked the beginning of resolut
primary pneumonia, but the extension was not succeeded by so defin
although a careful study of the temperature chart has convinced
crisis did, as a matter of fact, occur on the morning of the 23d and
fore the irregular temperature curve on the following days cannot b
as a manifestation of lysis.

CHAPTER XV

ACUTE FIBRINOUS PNEUMONIA—*Continued*

Physical Signs. Stage of Engorgement.—Both the duration and clinical findings of this period of the pneumonic process are uncertain. It may persist only a few hours or, as in a case observed by me in a lad of fifteen, it may not pass into that of demonstrable hepatization for a day and a half after the invasion. If the patient be examined, no matter how carefully, at this time there may be nothing more than the pain to indicate the seat of the process. This certainly holds true of the very commencement of this stage. As the engorgement increases, however, and approaches the stage of red hepatization pulmonary resonance over the affected lobe, most commonly at one or the other posterior base, becomes somewhat impaired and the breath-sounds are rather indefinite or possess a broncho-vesicular quality. If the pleura is also involved there may be friction over a limited area or there may be slight dullness. I have known more than one such case to be regarded as merely pleuritis until after physical signs of consolidation had grown distinct. The stage of engorgement passes gradually into the second stage and hence the signs of solidification develop insidiously. At one visit the results of examination may be uncertain and a few hours afterwards may be clear and unmistakable. On the whole, in the first stage of acute pneumonia the diagnosis has to be made rather by the history and symptoms than by the physical signs.

Stage of Red Hepatization. *Inspection.*—The patient's appearance is sometimes quite characteristic. His partly lateral decubitus, his bright eye, flushed face, perspiring skin and rapid breathing tell the story plainly. This is very apt to be the case with the sthenic type, whereas in other forms of the disease the clinical picture is indefinite.

The patient generally lies on the affected side because by so doing his pain is easier, while his dyspnœa is shown by rapidity rather than difficulty of breathing. Occasionally, however, the extent of the exudate or the associated bronchitis causes orthopnea; the nostrils dilate and the muscles of the neck contract strongly with each inspiration. Inspection of the chest detects restricted movement of the side on which the pneumonia is situated, and if a lower lobe is involved the base of that side may appear somewhat increased in size, though the intercostal spaces are not smoothed out unless pleuritic effusion is also present. The heart's impulse is not materially if at all displaced, though it may be more than normally diffused in cases in which the *processus lingualis* is solidified by the pneumonia (Osler).

Palpation.—Over the affected lobe pectoral fremitus is usually increased. This increase may be slight, however, when a pleuritic exudate intervenes. If the bronchioles are blocked by the pneumonic exudate (the so-called massive pneumonia), or if bronchitic secretions obstruct the transmission of vocal vibrations, pectoral fremitus may be absent altogether. Palpation may also detect enlargement of the spleen and the inferior hepatic margin below the costal arch. This is specially true in children in whom the organ becomes easily engorged as well as displaced.

Mensuration is rarely necessary, yet when resorted to fails to reveal important increase in the diameter of the base which to the eye may look larger than its fellow.

Percussion.—This means of investigation is of the utmost importance and should never be neglected. During the engorgement the results of percussion are at first very indefinite, but as this stage progresses and merges by degrees into the second, resonance over the involved lobe becomes impaired and assumes a slightly tympanitic quality (Figs. 24 and 25). This blending of dullness with tympany is due to the admixture of air and fluid in the alveoli (Gerhardt) and appears shortly before the note becomes distinctly dull from the complete filling of the lobules by the exudate.

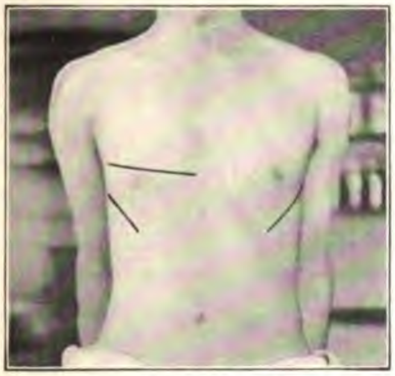


FIG. 24.—Location of interlobar fissures anteriorly.

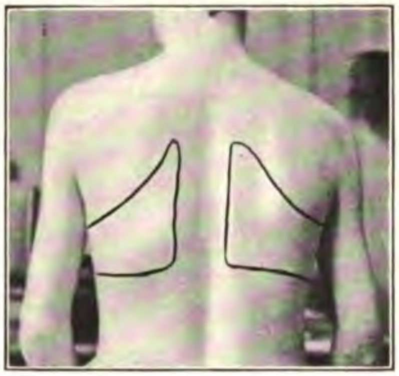


FIG. 25.—Location of interlobar fissures posteriorly.

In the second stage, that of red hepatization, pulmonary resonance is entirely lost over the affected part. The note is now intensely dull and there is a sense of great resistance. In typical cases dullness is found to extend throughout the whole lobe and to follow the line of the interlobar fissure. As the lower lobe is most often involved, and as this lobe lies chiefly behind, dullness is found to reach from just below the spinous ridge of the scapula all the way to the base, where it blends with that of the liver, and to pass obliquely forward and downward across the axillary region to terminate immediately below or a little within the nipple. The upper line of this dull area, moreover, remains stationary in whatever position the patient may lie, a differential point of great importance in doubtful cases.

Third Stage or Stage of Resolution.—No diminution of dullness may be perceived directly following the crisis, but very shortly, within the next twenty-four hours perhaps, the note grows less dull and for a short time again takes on a tympanitic quality. By degrees, in some cases with striking rapidity, resonance returns to the affected part and by the close of the second week percussion note becomes normal. In some instances however an appreciable impairment persists at the base, owing, probably, to the presence of adhesions from the pleuritis that attended the pneumonia.

Auscultation.—During the *first stage* the breath-sounds are enfeebled, but as hepatization proceeds they become progressively less vesicular until, by the time dullness has grown pronounced, they are distinctly bronchial. Indeed there is no affection in which this tubular quality of the respiratory murmur is so sharply defined, sounding precisely like that heard in health when the bell of the stethoscope is placed over the seventh cervical vertebra, which place, by the way, serves as a convenient point for comparison in studying the character of the breath-sounds. This bronchial character is often best perceived by the unaided ear. Now and then cases of pneumonia are encountered in which, notwithstanding exquisite dullness, the respiratory murmur cannot be distinctly or perhaps at all heard. This is owing to interference with the transmission of the breath-sounds from the trachea and large bronchi, by copious secretions, or by a massive exudate within the finer air-tubes.

There are also certain adventitious sounds in pneumonia which are very characteristic. These are the so-called *crepitant râles* which are a collection of minute crackling sounds having a peculiarly sharp distinctness and seeming to be produced immediately beneath the point at which auscultation is made. This râle, which, when once heard in its purity can never again be mistaken, is not to be detected throughout the entire course of pneumonia but only during a brief period just as engorgement is passing into complete hepatization. It may therefore be missed altogether if the patient is not seen and examined frequently at this time. Furthermore, the crepitant râle may not be audible during shallow breathing but only when the patient coughs or takes a deep breath. The râle then comes out distinctly at the end of the forced inspiration. It may be noted that the clicks making up this râle are all of exactly the same size and loudness and are not heard during expiration. Attention should be paid to these points lest this râle be mistaken for, or confounded with pleuritic friction or with the subcrepitant râle of bronchitis. This pneumonic crepitus is frequently obscured by friction sounds and by râles of associated pulmonary œdema and bronchitis. When such is the case a cough usually brings out the true crepitant click with distinctness amid the other moister sounds.

Dr. J. B. Leming of New York contended some years ago that this crepitant râle was produced within the pleural cavity and was therefore a pleuritic exudation râle. This contention finds supporters even now, and among them may be cited James Alexander Lindsay. It is true that a pleuritic friction may so closely simulate the crepitant râle as to be difficult of distinction; but in such an event it has always seemed to my ear that the individual clicks lacked the uniformity in size, sharpness and intensity possessed by the râle of acute pneumonia. I much prefer the view that this latter is generated within the alveoli or finest bronchioles.

This characteristic sound is lost during the height of the second stage, but reappears for a very short time during the fore part of the stage of resolution. It is however not precisely the same, being, as it seems to me, not quite so uniform in the characters previously described and heard in both expiration and inspiration. This *râle redux* is accompanied and may be partially obscured by subcrepitant râles which sometimes persist throughout the stage of resolution although growing gradually less and less numerous and distinct.

The breath-sounds which were plainly bronchial during the stage of red hepatization pass by degrees with the disappearing exudate through the bronchovesicular and finally into the vesicular quality of complete resolution.

Diagnosis.—In ordinary cases of sthenic pneumonia a correct diagnosis is not a matter of much difficulty. Even without an examination of the chest one may recognize the malady from the history of a chill followed by pain in the side, high fever, rapid respiration out of proportion to the pulse-rate, distressing cough and rusty, tenacious sputa. Considerable difficulty of diagnosis may arise if, on the other hand, the patient is seen early during the stage of engorgement or when chill and pain have not been marked; the respirations are not greatly increased in frequency and the expectoration is not bloody.

In the first few hours physical signs are apt to be obscure, namely, slight impairment of resonance and diminution of breath-sounds, and one may be in doubt whether the pain and cough be not due to pleurisy, especially if, as is not infrequent, a friction rub is audible. In such an event the physician must await development either of consolidation or of a pleuritic exudate as the case may be.

With hepatization of the lung appear increased vocal fremitus over the affected area, dullness, bronchial breathing and the characteristic crepitant râle, a group of signs which belong to no other affection than a pneumonic consolidation. Should these signs be circumscribed to a limited area at one base and the respirations be not materially out of proportion to the pulse, doubt may still exist; but if they occur in an upper lobe, they plainly indicate pneumonia. The nature of the pneumonia, whether fibrinous or tuberculous, may still be doubtful and hence the points of difference will be given under differential diagnosis.

In most instances a primary pneumonia may be correctly diagnosed by due attention to the history of sudden onset, to the loss of pulse-respiration rate and to other symptoms together with the conjunction of physical signs previously described. If the process is centric or its signs are masked by those of associated pleuritis, bronchitis or œdema, dependence must be placed on other means than examination of the lungs. Such difficulty, as a rule, only arises during the first three or four days of the process. The aids to diagnosis referred to are examinations of the blood, urine and sputum.

In hospitals and among physicians who have access to laboratory methods blood-smears may be made early and will generally reveal the presence of the diplococci. In the event of failure to detect the germ in this manner a blood-culture may be made when the nature of the case will be placed beyond doubt. Should meningitis attend and possibly disguise the lung affection lumbar puncture should be made and the fluid obtained be examined by smears or culture. It will generally reveal the pneumococcus.

A leucocyte count is valuable both diagnostically and prognostically. In no acute infection are the white blood-cells so greatly increased as in pneumonia and for this reason such examination is to be recommended. Nevertheless, that error in diagnosis is possible under such circumstances was proven by a case in Cook County Hospital of a woman whose blood, a few hours prior to death, showed a leucocytosis of 100,000. The autopsy disclosed acute miliary tuberculosis of the lungs with compact consolidation of the upper lobe which may have been fibrinous but could not be positively determined.

Practitioners who cannot for one reason or another resort to blood-examinations may derive valuable information by microscopic examination of the sputa. If properly stained and examined characteristic diplococci will be discovered. Such organisms may be derived from the mouth and on this account objection to this means of diagnosis is not unreasonable. Yet, if pneumococci are found in large numbers and especially if they are encapsulated, one may generally depend on their presence as diagnostic of pneumonia.

When a physician is deprived of sputum-examination as well as of blood-smears or cultures, then he may resort to urinalysis for aid in diagnosis. Should a marked reduction of the chlorides be determined, this fact, together with physical signs and symptoms suggestive of pneumonia, will generally serve to confirm the true nature of the disease. Herpes is of great significance and taken in conjunction with diminution in the chlorides and other symptoms, especially loss of pulse-respiration ratio, renders a diagnosis of pneumonia justifiable.

Differential Diagnosis.—Chronic or slowly developing disorders which give rise to dullness and bronchial breathing at either apex or base ought not to be mistaken for acute pneumonia if due attention is given to the anamnesis and symptoms. There are two chief affections that have to be considered, namely, pleurisy with effusion and acute tuberculous pneumonia of the apex. These conditions will be found in detail in the appropriate sections of this work and hence will be only briefly described in this place.

(1) *Acute Pleuritis.*—(*a*) This rarely begins with a single intense chill. (*b*) The sputum is seldom tinged with blood, but if so, from violence of cough, it is not rusty and is not so characteristically sticky. (*c*) The diameter of the affected side is increased, and this to a greater extent than is the case in lobar pneumonia uncomplicated by considerable pleuritic effusion. (*d*) There are signs of pressure on neighboring organs. (*e*) On palpation tactile fremitus is found diminished or absent. (*f*) The area of flatness has a curved upper border and shifts with change of patient's position unless the effusion is encysted. (*g*) Over the dull area breath-sounds are greatly diminished or wholly suppressed and voice-sounds are distant and feeble.

(*h*) If doubt still exists an exploratory puncture will probably settle the question.

The difficulties in the way of arriving at a diagnosis without such a procedure are illustrated by the case of Mr. W., seen in consultation with Dr. E. H. Webster of Evanston in May, 1899. The patient was a corpulent man of forty-five who, it was stated, had been taken ill a few days before with chill, pain in the right side and cough. When the doctor saw him not many hours thereafter he found a moderate fever, accelerated respirations and pulse, and on the right side dullness and bronchial breath-sounds which he took to be pneumonic. Dr. Arthur Edwards had been asked to see the man and had concurred in the diagnosis of acute pneumonia.

Three days later, on May 19th, I saw the case and made out the following physical signs. The patient lay on his back but turned a little to the right. In other respects inspection was negative. Palpation showed decreased pectoral fremitus at the right side; the lower border of the liver could be felt at the level of the umbilicus and was not tender. Percussion disclosed marked dullness over the entire right half of the thorax, with exception of the area occupied by the middle lobe which was relatively more resonant, though not so resonant as was the left lung. It was particularly noted that the upper lobe was just as dull as the lower. Over this entire dull region breath- and voice-sounds were absent, while over the middle lobe they were feebly heard. The left lung was resonant, and it was specially observed that the heart was not displaced, as appeared to be the case with the liver. This state of the latter organ suggested the expediency of an exploratory puncture, but the suggestion was not acted on by the attending physician because of the belief that the case was one of pneumonia and the determination of the presence of a pleuritic effusion was thought to be a matter of no very great importance.

The physical signs were not clearly those either of a pneumonia or a pleurisy with effusion, and made the differential diagnosis not easy. If it was the latter, as suggested by the absence of voice- and breath-sounds and the apparent downward displacement of the liver, then why was the upper lobe more dull than the middle and why was the heart not pushed to the left as certainly ought to be the case with so massive an accumulation of fluid as would be necessary to produce intense dullness over the upper lobe. On the other hand if this were a pneumonia, then how account for the absence of breath-sounds and voice-sounds, and for the lowered position of the inferior hepatic margin. On the whole, therefore, it was concluded that this was a case of acute pneumonia of the kind known as massive, the bronchioles being so blocked by the exudate as to prevent the transmission of both voice-sounds and respiratory murmur.

Moreover, the mode of onset which it was stated had been with a chill as well as pain in the side, and the perverted pulse-respiration ratio seemed to support this view. Finally, the examination made of the sputum at the Columbus Medical Laboratory showed pneumococci in abundance. Lastly, the examination of the blood made by Dr. W. A. Evans a number of days later with a view to determining the amount of leucocytosis disclosed 21,990 white cells. In view of all these facts we entertained no doubt of the existence of pneumonia in this case. I saw it several times between May 19th and 23d, and at my last visit noted that the middle lobe had also become dull, which circumstance was attributed to an extension of the process.

Our surprise and disappointment can be imagined, therefore, when, at the autopsy, May 30th, it was discovered that the condition had never been a pneumonia but was a pleuritic exudation which, by reason of old adhesions, had been circumscribed and led to the peculiar signs described. Condensed from the necropsy records made at the time the condition was as follows.

The left lung was negative with exception of congestion and a few old adhesions in the interlobar fissure and over the diaphragm. The right lung was bound by old tough adhesions to the anterior chest-wall from apex to base and as far outward as the vertical nipple line. Adhesions also united the inferior surface of the lower lobe to the diaphragm and also extended up the back close to the mediastinum nearly to the extreme apex. In other words, the only part of the right lung not adherent to the parietes was that portion of the lower and upper lobes lying between the adhesions along the inner borders front and rear and those at the apex and on its inferior surface, that is, its postero-lateral aspect. The visceral pleura was thickened and the lung collapsed so far as the adhesions permitted. The heart was in its normal situation and the liver was enlarged and fatty but not displaced, the pleuritic adhesions having also prevented that.

In consequence of the peculiar arrangement of these old fibrous bands the pleuritic exudate had been forced upward toward the apex and had neither been able to push the liver down nor, by extending forward of the nipple, to force the heart over to the left. It was thus easy to see why the upper lobe had given so dull a percussion-note. Not only was this collapsed, but the effusion occupied a much higher position posteriorly than it would have done had there been no adhesions to prevent it from gravitating to the bottom of the thoracic cavity.

In explanation of the leucocytosis and the more marked elevation of temperature toward the end of the illness, it may be stated that the exudate was found to consist of thick flaky pus and was therefore an empyema. This was not submitted to bacteriological examination but, to judge from its richness in fibrin and the presence in the sputa of great numbers of pneumococci, it was probably due to these organisms.

It is interesting to speculate also as to whether this was an acute pleurisy or was a secondary infection of an old-standing effusion. The adhesions appeared to date from a pleuritic attack twenty years back, but the man's history just prior to his fatal illness and Dr. Webster's familiarity with the conditions of his chest make it quite certain that any considerable amount of fluid had not previously existed.

So grave an error could have been corrected had we only made an exploratory puncture. In any other case in which physical signs point to a lobar pneumonia and the absence of breath-sounds and vocal fremitus suggests a massive exudate blocking up the smaller air-tubes I shall certainly introduce a needle. Performed under proper aseptic precautions it can do no harm and may be productive of great good.

(2) *Acute tuberculous pneumonia of the apex* is the other affection which is likely to be mistaken for acute croupous pneumonia and therefore merits special consideration as regards differential diagnosis. These cases are instances of acute confluent broncho-pneumonia of tuberculous origin pure and simple. The process may involve the entire upper lobe, and when it does its similarity to a fibrinous pneumonia of the apex is so close as to make a differential diagnosis impossible in the early stage of the process. The following are the main points both of similarity and contrast in the two affections.

(*a*) In both the onset is abrupt but in the tuberculous form an initial chill is likely to be less pronounced. (*b*) Pain in the affected side is common to both and in both there are cough and rapid rise of temperature. (*c*) In tuberculosis an initial hæmoptysis is much more common and the sputum is apt to be more distinctly bloody, not rusty. (*d*) Temperature in both is high but in the tuberculous form the fever is apt to show a tendency to irregularity and is therefore not so typically continuous (Powell). (*e*) Pulse and respiration are accelerated but the former is apt to be more rapid in proportion to the breathing than in the acute croupous form. (*f*) Crisis does not occur in the tuberculous pneumonia, and with the signs of softening the lung does not display evidence of clearing up day by day as in the acute lobar form. (*g*) With the crepitant râles marking the stage of softening there are also heard, here and there, larger clicks which mark the areas where acute destruction of pulmonary tissue is beginning to take place. (*h*) Expectoration rapidly grows abundant and purulent and may contain elastic fibers as well as tubercle bacilli. (*i*) The fever assumes the hectic type as time proceeds and emaciation and profuse sweating accompany this type of pyrexia.

In most cases an early differentiation is absolutely impossible and hence the disease is generally considered an ordinary acute pneumonia of the apex until the subsequent progress of the affection establishes its true nature. I recall a case of this kind in my service at Cook County Hospital. The patient was a young man of about twenty-five who, when admitted, showed the signs of pneumonia of the left upper lobe. The history was of sudden onset with pain in the left side, cough and headache. The left cheek presented a bright red spot, respiration and pulse were both increased in frequency, and the temperature averaged high. The sputum was bloody so that with pronounced dullness and exquisite bronchial breathing over the left lobe, the diagnosis was plain of an acute apex pneumonia.

Apparently it was an ordinary lobar inflammation, but crisis did not take place after the usual length of time, although moist râles began to appear below the clavicle and expectoration became distinctly more fluid and purulent. The râles were fine and sticky and brought out with great distinctness and in immense numbers with each act of coughing. Fever now began to decline and it was thought the case was going to terminate by lysis.

As days went on, however, coarser clicks were detected amidst the fine crepitations, the temperature remitted and even intermitted in the morning but rose to 102° or even 104° F. in the afternoon, perspiration grew quite pronounced, the patient emaciated perceptibly, lost strength and continued to cough and expectorate freely. The respirations sank to twenty-eight or less but the pulse-rate remained high and the other characters of tension and volume corresponded with the weakness of the patient. Examination of the sputa now revealed the presence of tubercle bacilli, and it became plain that instead of an acute croupous pneumonia of pneumococcus origin we had to do with an acute pneumonic phthisis.

The case was presented to my class together with another right apex pneumonia which was clearly in the stage of resolution, and the contrast between the two was very marked. Both had commenced very similarly and for a time both had presented very much the same clinical course as regards the local and constitutional disturbance, excepting that the patient with the true fibrinous pneumonia had for a short period displayed acute delirium. In this one crisis took place, and the upper lobe cleared up rapidly while in the other the course became that of active lung-destruction with hectic temperature and other signs of mixed infection.

In the diagnosis of croupous pneumonia of healthy adults one has rarely to differentiate from other conditions than those already mentioned, but one may often be called upon to detect the disease when masked by other affections. When this is the case the pneumonia does not occur as the frank or sthenic form but is atypical and the reader is referred to that variety for a further consideration of this subject. In a tuberculous apex-pneumonia and any other affection suggesting fibrinous pneumonia, modern methods of blood examination ought to settle the question.

It occasionally happens that a genuine lobar pneumonia is masked by a meningitis of the same bacterial origin, in which event the ordinary subjective manifestations of cough, sputum, etc., are concealed and one must depend upon physical signs and blood cultures for a diagnosis. Such a case was seen recently at the Cook County Hospital. A man of apparently middle age was brought in with the history of having been treated by an unknown physician for meningitis.

The man was unconscious, with dilated pupils, cold clammy skin, a very feeble moderately accelerated pulse and stertorous breathing. An attempt to flex the neck was resisted by marked muscular contraction and to judge from his moaning caused pain. Likewise an attempt to extend the leg while the thigh was at right angle to the trunk was found impossible on account of strong contraction of the flexor muscles (Kernig's sign).

Satisfied regarding the existence of a meningitis, we next turned our attention to the chest and discovered dullness, bronchial breathing and subcrepitant and a few crepitant râles in the left lower lobe behind. Before the rectal temperature could be taken it was discovered that he had been brought to Ward 10 by mistake and the patient was carried off to the service of the homœopaths.

He died, I was informed, a few hours later, but was not subjected to a *post-mortem* examination, so that it was impossible for us to have our diagnosis confirmed either by a more extended examination or by a necropsy. It seemed tolerably certain, however, that the man was suffering from a general pneumococcus infection of which, according to the history, only the meningeal inflammation had been recognized. The occurrence of such a meningitis will be further considered under complications.

Complications.—Under this head must be considered (1) inflammations of other structures which are due to the same cause (the so-called specific complications), and (2) preëxisting diseases of other organs (the accidental complications).

It is not yet definitely settled whether the complications of the first group are to be regarded as but different manifestations of a general pneumococcus infection along with the pneumonia, or as secondary inflammations in consequence of the bacteria having been carried to the part in the blood- or lymph-stream. The latter view would seem to be supported by the observation that most of the specific complications occur during the height of or immediately after the pneumonia. In the case of meningitis the microorganisms may, as suggested by Liebermeister, gain access to the brain from the nasal passages by the way of the ethmoid.

Pleurisy is by all odds the most common complication of lobar pneumonia. The pleura shares in the inflammation whenever the pneumonic process reaches the periphery, but it merits the term of complication only when it leads to a considerable and recognizable effusion, at least 400 c.c. of fluid. In such an event the case may be quite properly termed a pleuro-pneumonia, and this appellation should not as is so often done be given to cases characterized by intense pain.

Statistics vary within rather wide limits as regards the frequency of this complication. Thus, at Vienna, of 5,738 cases of pneumonia pleurisy was discovered 298 times or 5.2 per cent; at Stockholm (Huss), 2,616 cases, pleurisy, 104 or 4 per cent; at Basle (Fismer), 230 cases, pleurisy thirty-five, or 15.3 per cent; Aufrecht, 1,501 cases, pleurisy eighty-nine or 5.5 per cent; Sello, 750 cases, pleurisy sixty-five or 8.7 per cent; Grisolle, 12.6 per cent.

When the pleuritic involvement is sufficient to be dignified by the term complication, the exudate may be sero-fibrinous or purulent. In the former there is always a remarkable richness in fibrin which is speedily deposited in thick flakes or layers. If the effusion is copious enough to occasion demonstrable pressure upon adjacent viscera, as heart and liver, it is to be recognized by these together with other signs indicative of pleurisy with effusion.

The pleuritis may anticipate, accompany or follow the pneumonia, but the last is by far the most usual. Of fifty-seven cases in which Sello demonstrated the presence of fluid by puncture (Preble) only two occurred at the onset, thirty-one during and twenty-four after the pneumonia. It is said to make its appearance generally on the fourth or fifth day of the pneumonia, and to exert but small effect on the course of the pneumonia. According to Huss the mortality may be increased only 0.8 per cent.

Empyema.—The frequency of this complication is shown by statistics to vary considerably. Aufrecht found twenty-three empyemas among 1,501 pneumonias, 1.5 per cent; Sello, thirty-four among 750 cases, 4.5 per cent; Sears and Larrabee, ten in 949 cases, 1.5 per cent; Norris, six in 500 cases, 1.2 per cent; Meyer, twelve in 500 cases, 2.4 per cent; total of ninety-four empyemas out of 4,200 cases of pneumonia or 2.4 per cent (Preble). This form of pleuritic exudate may be abundant, especially in children, but is usually small in amount and circumscribed because of its richness in fibrin. When occurring on the same side as the pneumonia it is difficult or impossible of recognition without exploratory puncture during the height of the disease. The physical signs by which empyema may be recognized will be found detailed in the chapter on Acute Pleurisy. It is this complication that is often responsible for the continuance of pyrexia and other symptoms after the resolution of the pneumonia.

Pericarditis.—This complication undoubtedly exists more frequently than it is recognized at the bedside. This is due to many circumstances, but mainly to the fact of its auscultatory signs being obscured by those of the lung condition, as well as of failure on the part of the physician to search for it systematically. There is considerable discrepancy between clinical and *post-mortem* statistics regarding its frequency. Thus from figures given by Preble the range in its bedside recognition is from 0.3 to 3.9 per cent while between Osler's statistics and those of Kerr there is a range from 5 per cent to 15.8 per cent.

The relation of the pericarditis to the situation and extent of the lung involvement is shown by Preble's figures taken from the *post-mortem* records of Cook County Hospital and published in his paper on The Ætiology of Pericarditis, 1901. There were studied seventy-three instances of this complication and in fifty-five of these the lobes were particularly mentioned. Pneumonia of the right lung was present in twenty-three, of the left lung in twenty-one and of both lungs in eleven. The liability to pericarditis is especially great in pneumonia of the right, middle and left upper lobes, particularly if the *processus lingualis* is involved. It stands in intimate relation also to the number of lobes affected. The invasion of the pericardium may be by direct extension or by the blood-stream rather than by the lymphatics.

Endocarditis.—This is another complication which occurs more often than its clinical recognition would indicate. The reason therefor lies in the fact that in fatal cases of pneumonia which furnish *post-mortem* evidence of valvulitis time is not allowed for clinical signs of the complication to declare themselves. The frequency of endocarditis in this affection varies within wide limits, from 0.06 per cent to 16 per cent, but of a total of 11,243 collected cases there were 126 of endocarditis, i. e., 1.1 per cent, while of 1,775 pneumonias that came to autopsy there were eighty-six instances of endocarditis, 4.28 per cent.

The endocarditis is of the malignant variety more often than is generally supposed. Thus, of 209 cases of septic endocarditis collected by Osler from the literature fifty-four or 25.8 per cent were in pneumonia, while Canthak found an antecedent pneumonia in 14.2 per cent of cases of infective endocarditis. The pneumococcus has been identified in the thrombotic masses on the inflamed valves. The semilunar valves are involved more often than are the auriculo-ventricular and in one seventh of Netter's collected cases the lesion was in the right heart. The mortality is very high, more than one half of the cases dying inside the first month (Preble).

Meningitis is a very serious complication since the cases usually prove fatal in from a few hours to three or four days after its onset. It usually develops during the height of the pneumonia and yet it may precede the latter. The frequency of this complication varies much as shown by statistics. Osler found it in 8 per cent of his fatal cases, while Sello discovered it but five times in 750 cases, 0.6 per cent. Aufrecht found it in ten out of 1,500 cases, also 0.6 per cent, while at Basle it was found in three out of 230 or 1.3 per cent.

The frequency of meningitis is greater in some epidemics than others, but the localization of the pneumonia does not appear to determine the development of this complication. Neither does it seem to depend upon the extent of lung involved since it is not more common in double than in single pneumonias (Preble). For some unknown reason it appears to be rather more common in males than females and to occur somewhat more often between forty and fifty than at other ages.

The inflammation may be limited to the cerebral meninges, or it may involve the coverings of the cord as well as the brain. The exudate may be sero-fibrinous or purulent. The meningitis is said to be associated with pericarditis in about one third of the cases.

The diagnosis of meningitis, particularly in the early stage, is often very difficult and for that reason it cannot always be differentiated from simple irritation of the brain. Kernig's sign is of value in its detection, but in doubtful cases resort may be had to Quincke's lumbar puncture and bacteriological examination of the fluid thus obtained.

Hemiplegia, or other paralysis, is also mentioned by Liebermeister as sometimes occurring either in consequence of compression by the meningitic exudate or as a result of embolism, thrombosis or hæmorrhagic effusion. Such paralyses may be permanent or transient according to their cause.

Bronchitis, more or less localized, accompanies most cases of fibrinous pneumonia, but occasionally this becomes so extensive in the opposite as well as in the pneumonic lung as to prove a formidable complication. In such a case the bronchitis is a manifestation of diplococcus action and the secretion may be purulent or fibrinous. The blocking of the tubes by fibrinous plugs or secretions may be so extensive as seriously to threaten life. I recall such a case in which the hypodermic administration of large doses of apomorphine failed to excite emesis and empty the tubes.

Otitis media is among the less common complications and results from invasion of the middle ear by the pneumococci through the Eustachian canal. It is not likely seriously to influence the course of the disease, and yet in young children may, by pressure, occasion alarming symptoms, as vomiting and even convulsions (Ball), which latter may suggest meningitis.

Icterus as a complication of croupous pneumonia probably occurs far more often than is recognized or stated to be the case by most writers. It was mentioned by Hippocrates and other ancient authors who attributed pneumonia to alterations in the secretion and elimination of the bile. The frequency with which jaundice occurs varies greatly, being undoubtedly more prevalent in some epidemics than others. Traube thought it occurred more often in the summer than the winter months, but this view does not seem to be entertained by modern clinicians. The wide variation seen in statistics (0.6 for Vienna to 28.3 for Basle) probably depends largely on the care with which this complication is sought for, since in its slighter grades icterus may be readily overlooked.

Equally diverse opinions appear to be held regarding its causation, and Pétrow even went so far as to regard jaundice as an accidental complication, there having been in all his thirteen cases a lesion of the biliary passages with duodenitis in eight, angiocholitis in five and hypertrophic cirrhosis of the liver in two. Some other explanations may be equally well dismissed as untenable, e. g., compression of the finer bile ducts by hepatic stasis, absorption of bile because of inhibited movements of the diaphragm, hæmolysis through destructive action of the diplococci, formation and absorption of bile pigments during the stage of resolution. The icterus is more likely due to inflammatory obstruction of the biliary ducts or is the form known as hæmatogenous.

In its milder grades this complication may not affect the course of the pneumonia, but when intense it may unfavorably affect the brain and nervous system, even producing a comatose condition in some instances. In other cases it may excite delirium or blunt the patient's sensibility. Its effect on the heart is, according to French writers, to induce dilatation of the right heart, as is seen in cases of gall-stones, and Preble calls attention to the harm that may be wrought in pneumonia by its thus intensifying the right ventricle-dilatation already caused by the lung involvement. Intense jaundice may also conduce to the development of albuminuria or even acute nephritis.

The icterus also injuriously influences the nutrition of the pneumonic patient by its effect on the gastro-intestinal tract. It is also worthy of note that in these cases of pneumonia the sputa are bile stained, even to the extent of being of a greenish color, whereas the saliva and mucous secretions are not (Liebermeister). Cases which exhibit this complication to a marked degree were formerly designated as Bilious Pneumonia.

Arthritis has now come to be a more frequent complication of pneumonia than was supposed prior to the monographs of Leroux (1899), Cave (1901) and Herrick (1902). The last named was able to add twenty-one cases to those collected by Leroux and Cave which made a total of fifty-two cases in the literature up to the date of Herrick's excellent paper from which I have freely drawn.

The reason this complication has not been more often recognized is probably to be found in the fact that the arthritis was thought to be either rheumatic or pyæmic. It is to be distinguished from such, however, by its monarticular localization, its permanent rather than shifting character, the presence of fluid and the absence of other earmarks of rheumatism. Were other proof of the specific nature of this form of arthritis needed, it would be found in the identification of the pneumococcus in the joint exudate, as happened in all of the cases collected by Herrick.

The articulations most frequently affected are those of the upper extremities, twenty-three out of the fifty-two cases. In eighteen the joints of the lower extremities alone were involved. In eleven both upper and lower extremities were affected. The joints most often attacked were as follows: Knees twenty-five times out of a total of eighty-four joints, shoulder twelve times, elbow nine, sterno-clavicular and wrist each eight, hip, ankle and metatarso-phalangeal each three, metacarpo-phalangeal twice. In thirty-two instances or 61.5 per cent the arthritis was limited to a single joint, as follows: shoulder ten times, knee nine, wrist five, elbow two, sterno-clavicular four and hip, ankle, metacarpo-phalangeal and metatarso-phalangeal each one time, while of the remaining cases two articulations were attacked twice, three joints four times, four joints once, and more than four joints three times. It is thus apparent that with the exception of the sterno-clavicular articulation the larger joints are the ones most often inflamed.

The arthritis may occur before the pneumonia, as was observed five times, during the course of the lung affection, or not until after the crisis has passed and the lung has cleared up. This last fact is one of very great importance and may explain why the arthritis is so likely to be considered rheumatic. It is also worthy of note that previous injury to the joint and rheumatism appear to act as predisposing causes. The changes which take place in the joint depend upon the duration of the inflammation as well as upon the virulence of the germ and the resisting power of the individual.

In acute cases there is an ordinary synovitis with exudation that may be serous, sero-fibrinous or purulent, the pus being thick and creamy as is characteristic of pneumococcus inflammation. In subacute and chronic cases the changes may be very destructive, erosion of the cartilages, inflammation of the bones, destruction of the ligaments and even involvement of the muscles and other structures about the joint showing a periarthritis along with inflammation of the articulation proper.

The joint complication may escape recognition if it occurs during the acme of the pneumonia, especially if the arthritis develops gradually and the joint symptoms are slight. Its occurrence during convalescence is ordinarily attended by local symptoms which render its recognition easy. Pain in the joint may be intense and abrupt in onset or may develop slowly and be slight. There is more or less tenderness which, when the periarticular structures are involved, is superficial and particularly pronounced. Redness, swelling and even fluctuation may occur, and œdema of the external tissues is present if the inflammation attacks the structures round about the joint. In a word, the clinical manifestations are those of arthritis from any other cause.

Other infrequent complications are parotitis, severe pharyngitis, laryngitis and it may be œdema glottidis, peritonitis said by Osler to be exceedingly rare, mediastinal abscess and croupous inflammation of the gastro-intestinal tract. Diarrhœa, occasionally observed, apparently independent of actual inflammation, may be so severe as to prove a serious complication. It may be fœtid in character and attended with great abdominal distention. Such a diarrhœa is probably a manifestation of something more than a mere putrefactive decomposition of the ingesta, although I have observed it in a case after the crisis when it seemed to be a result of fermentation of the highly albuminous dietary and yielded after such articles of the food had been replaced by articles of nonanimal origin.

Acute nephritis is not a very infrequent occurrence in pneumonia which may be overlooked if daily examinations of the urine are not made. It may prove a serious complication, yet, according to Liebermeister, does not often eventuate in chronic renal disease. It is especially liable to occur in persons whose kidneys were previously not healthy, and hence in old people. With a view to the possible prevention of this complication patients should be encouraged to keep the urine diluted by the ingestion of large amounts of fluid.

Pulmonary abscess, said by some to be rather more likely in apex pneumonias, is also an infrequent complication, having been found in 4 per cent of Osler's autopsies and 1.2 per cent of Aufrecht's 253 cases. It may be of large size but is usually so small as to make the detection of its site difficult or impossible, even when the characteristic purulent sputa with hæmatoidin crystals make their appearance. The pneumonia generally clears up while the abscess persists, and convalescence is tedious or the patient finally succumbs to exhaustion and sepsis.

Gangrene of the lung is still more uncommon, Sello having found but three instances in 750 pneumonias and Aufrecht none in 1,501 cases. It may be very small or extensive, but in either event can only be diagnosed by the characteristic stinking sputa.

Duration and Termination.—As so tersely expressed by Aufrecht, a pneumonia may last from one day to three weeks. Formerly I was skeptical and even sarcastic in my criticism of those who believed in the so-called abortive forms since the data on which the diagnosis was based seemed to me insufficient. With advancing years I have grown more charitable, and although having seen but one case which might possibly come under this head, I am willing to accept the statements of such clinicians as Leube, Weil, Thomas, Tophoff, Baruch and Bernhard as convincing.

Last year a case was observed in Chicago by Drs. F. S. Churchill and J. L. Miller which gave the signs of acute croupous pneumonia even to characteristic rusty sputum and yet showed signs of resolution within twenty-four hours. How such a state of things can take place, whether the case passes with incredible celerity through all three stages or is aborted in the first stage can only be a matter for conjecture. The fact must be accepted on faith by such of us as have not been convinced by personal experience.

The long duration to three or more weeks is to be explained in several ways. Repeated extensions or even the rare relapses may retard convalescence, or the case may end by lysis, the temperature and other general and local symptoms disappearing by slow degrees. In other cases the course of the disease may be protracted by complications or by delayed resolution.

This last-mentioned possibility furnishes a fruitful theme for discussion by writers. It was taught by earlier authors in particular, that instead of resolution taking place within a few days or a week, as is usual, it might be retarded by different not altogether understood factors, among which influences are old age and general feebleness.

The opinion now seems to be gaining ground, however, that the persistence of dullness, bronchial breathing and even of crepitant râles is to be attributed not to slow absorption of the exudate but to its conversion into fibrous tissue. In other words the exudate undergoes organization and that portion of the lung becomes permanently indurated. (See Morbid Anatomy.) Fraenkel goes so far as to state that he has practically abandoned his old notion based on the teachings of Charcot that the persistence of local signs is due to slow resolution, and he believes, from his study of twelve such cases that came to autopsy, that the condition is one of organization. Such resulting induration may be very limited or may involve the greater part of the lobe occupied by the exudate. It is such cases that become instances of chronic pneumonia with consequent dilatation of the bronchi. They may even lead to retraction of the lung and to chest-deformity, as witness a case observed by Stokes and cited by Fraenkel.

As already stated the retention of local signs denoting a failure on the part of the lung to clear up is seen most often in aged and debilitated subjects. Yet now and then they may persist in the young and robust. When such is the case the individual loses his constitutional symptoms, convalesces, though somewhat tardily, and after a time is able to resume his former occupation. He may experience slight shortness of breath on extraordinary exertion and may complain of pain in the affected side.

Dullness and diminished bronchial breathing are found at one base and voice-sounds and fremitus are somewhat increased, thus demonstrating that the local signs are not owing to pleuritic thickening. After the lapse of many months or even years the percussion-note assumes a more resonant quality, showing that resolution has at length proceeded or that the areas of limited organization have become disguised by compensatory emphysema.

In my hospital service in the Winter of 1905 there was admitted an old man who gave his age as a hundred. He certainly looked to be that old and when he was discovered to have an acute croupous pneumonia of the right lower lobe it was believed he would quickly succumb. His fever was high for so aged a person, averaging not far from 103, respirations were frequent and labored, the pulse scarcely appreciable in the beady, wirelike artery, the heart-sounds rapid and arrhythmic and obscured by the fine and coarse bubbling râles that filled both lungs, while over the entire right lower lobe was well-marked dullness. Crepitant and subcrepitant râles largely concealed the breath-sounds which, however, could be made out to be feebly tubular.

Cough was infrequent and expectoration was absent or swallowed. Low muttering delirium was present and yet the patient could be aroused and made to answer questions. Urine and fæces were passed involuntarily. The toxæmia was pronounced and yet the patient held out surprisingly. Death at length proved conqueror and at autopsy the findings were as follows:

1. Lobar pneumonia in the stage of organization in the posterior part of the right lower lobe and the whole of the right middle lobe.
2. Acute tracheo-bronchial lymphadenitis.
3. Œdema of the anterior portion of the right lower lobe.
4. Atrophic emphysema of the right upper and the whole of the left lung.
5. Bilateral fibrous pleuritis, with calcification of the adhesions on the right side (Fig. 26).
6. Chronic aortic and mitral endocarditis.
7. Healed anæmic infarcts of the spleen.
8. Marked atheroma with calcification of the entire aorta except the ascending portion of the arch.
9. General arteriosclerosis.
10. Arteriosclerotic atrophy of the kidneys.
11. Chronic interstitial myocarditis.
12. Slight interstitial hepatitis.

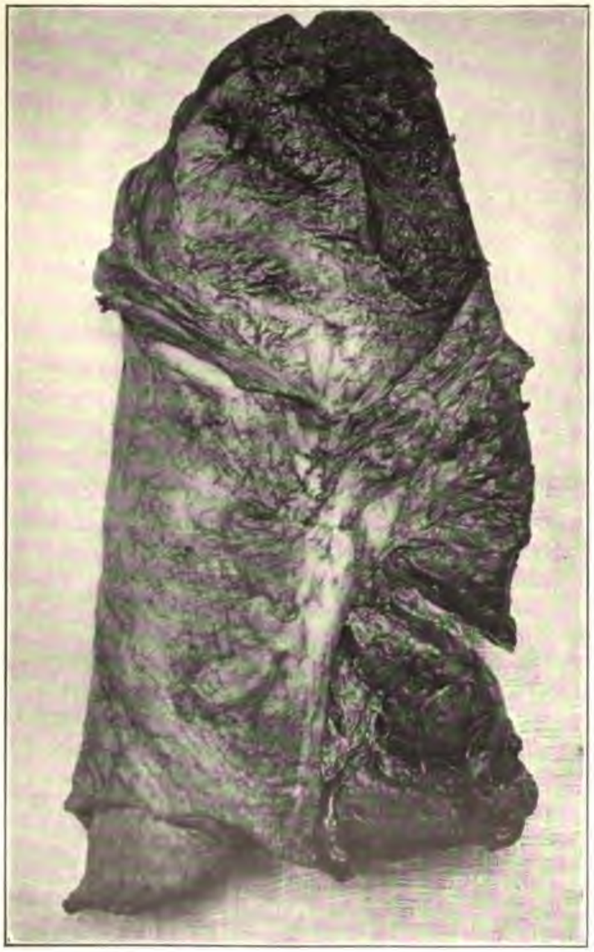


FIG. 26.—Calcification of old pleuritic adhesions in case narrated on page 249.

The affected portions of the right lung were very firm and elastic, not collapsing in the least on removal from the thorax, and resisting indentation by the fingers in a manner contrasting sharply with the œdematous or emphysematous lung about it. On section it showed a pinkish-gray, distinctly translucent surface much mottled by anthracotic pigment. The surface was dry and only from the larger blood-vessels was fluid obtained on pressure. Crepitation was very feeble but enough air was present to float the small portion cut off and tested in water. Microscopically (Plate V), the fibrin of the pneumonic exudate was represented by occasional masses of hyaline material found in the alveoli. For the most part however the alveoli were filled by a newly forming connective tissue. Delicate capillaries were into the air spaces, surrounded by leucocytes and embryonic con cells, which absorbed and replaced the fibrin and ultimately led eration of the air-cell.

Another, fortunately not very frequent, mode of termination or gangrene. These have been considered under Complications a separate chapter devoted to the subject, and to these the read for particulars.

CHAPTER XVI

ACUTE FIBRINOUS PNEUMONIA—*Continued*

Clinical Varieties. Atypical Pneumonias.—If the student expects to find all his pneumonias corresponding to the classical picture as described in the foregoing pages, he will be doomed to disappointment. In private practice among the well-to-do, or at least such persons as enjoy a healthful environment, he will find the majority of his cases sufficiently typical to render easy their recognition as lobar and due to the pneumococcus. Nevertheless, even here examples will now and then be encountered of what are generally spoken of as *Atypical pneumonias.*

In hospital practice and in unsanitary surroundings it is very common to see deviations from the characteristic type, which prove diagnostically puzzling and perplex the practitioner as to the ultimate outcome of the process. Such cases also stimulate doubt in the mind of the observer concerning the invariable presence of the diplococcus of Fraenkel as the sole or even the main causative agent of the pneumonia.

The perplexity seems to be enhanced by the results of sputum examination, since this generally shows a variety of germs. Pneumococci are seen, and in addition streptococci and often influenza bacilli, these mixed germs often being in far greater number. In such an event one is naturally tempted to look upon the case as an example of mixed infection, and I have seen cases in which I expressed such an opinion. This was especially so in an atypical pneumonia seen in consultation at Macomb, Ill., which, developing after several days of *malaise* with bronchitis and other symptoms seeming to indicate the grippe, displayed consolidation of a patchy distribution. Pain was a particularly pronounced feature and the temperature was erratic. The sputa were not rusty but were tenacious and on microscopic examination revealed influenza bacilli in great excess over diplococci. Recovery took place by lysis.

It is probable that in this and similar cases the pneumococcus is really the causative agent and that the other germs are derived from the fauces or have supplanted the diplococci. The fact that these latter, owing to their vulnerability, frequently fail of demonstration in the late stages or after crisis and may then be replaced by other organisms, renders it unsafe to conclude that they were absent altogether or were blended with other bacteria. In corroboration of this statement may be cited the well-known bacteriological investigations of the Klemperer Brothers whose work Fraenkel declares he has amply verified.

The Klemperers aspirated the lungs in a number of cases of pneumonia close to the periphery of the infiltration, and without exception were able by appropriate culture and inoculation in rabbits to demonstrate only the pneumococcus. In one of their cases the fluid withdrawn three days before the crisis contained this organism in numbers, whereas eight days later, and after the death of the patient, only streptococci could be discovered. In the light of such facts, therefore, it appears unsafe to assume that when a croupous pneumonia deviates from the usual type it does so because of some other than a pneumococcus infection or of a mixed infection. Moreover, the whole subject seems to me simplified by accepting the ætiological unity of the disease and by regarding clinical variations as due to peculiarities residing either in the diplococcus or in the individual afflicted. It is certainly in accord with bacteriological facts to assume marked differences in the virulence of the germ in different cases, which may account for some of the clinical peculiarities.

Experience also forces us to the conclusion that deviations from the ordinary type are determined by individual factors, in particular by age, habits and previous disease. Nevertheless, when all such conditions are granted, there still remain cases in which the atypical manifestations are inexplicable on any yet established hypothesis and we must simply accept them as clinical facts without attempting an explanation.

Migratory Pneumonia (*Pneumonia migrans, Creeping Pneumonia*).—This form of the disease is characterized by the fresh invasion of other portions of the lung than that first affected as shown by the usual signs of dullness, bronchial breathing and crepitant râles. *It is essential that the area or areas previously involved manifest objective evidence of progressing resolution while neighboring parts are becoming attacked.* One not infrequently hears this condition of things spoken of as an *extension* of the pneumonia. Such is not strictly accurate, however, since in typical cases of genuine fibrinous pneumonia, as remarked by Fraenkel, it is not uncommon for the infiltration to begin in a limited area and thence extend throughout the lobe without any sign of resolution in the primary zone, thus constituting a true extension.

Wunderlich designated the form now under consideration *erratic pneumonia* and Fraenkel adopts the same term. Friedreich likened it in its progressive tendency to erysipelas, emphasizing, as did Homburger, the invasion by continuity of areas contiguous to the part previously involved. In this sense it is a creeping pneumonia, whereas Wunderlich and Fraenkel prefer to restrict the term migratory to cases in which zones of later development are separated more or less widely from the original seat of the disease.

As a matter of fact the distinction does not appear to me an essential one, provided it be borne in mind that one area must be clearing up while another is being attacked. In this manner an entire lobe or indeed a whole lung may show successive stages of involvement. Moreover, it is not necessary for the migrating process to be restricted to only one lung, since it may invade the opposite one in the same manner.

As might be expected, these recurring manifestations of lung implication exert a marked influence over the course and prognosis of the disease. Friedreich dwelt on the typhoid character of these cases. Such is not always true, however, since this type of pneumonia is often distinguished by nothing more than irregularity of the temperature curve. Authors generally describe the temperature as remittent or intermittent, the remissions agreeing with the occurrence of resolution in the several areas and the exacerbations with the fresh inflammatory action.

Wilson Fox states that even recurring chills may be noted, bespeaking new zones of pneumonic consolidation. This was strikingly illustrated in a case of senile pneumonia, three chills occurring inside of thirty-two hours. Although the fever-curve may display such an irregular character, it is not invariably so, but may remain continuously high, the same as in ordinary cases. Thus Fraenkel states that in twelve of Ruge's collected instances of migratory pneumonia the fever was a continuous one. Yet, if the character of the pyrexia is not especially influenced, its duration is. The disease is unavoidably protracted and may drag along for twelve days (Homburger), seventeen days (Ruge), and even six weeks (Fox).

In other respects there may be nothing peculiar in the symptomatology. Sputa may or may not be rusty, and cough may likewise show considerable differences. In the Winter of 1903 I attended an old negro servant in my own household who ultimately died of this form of pneumonia. The process began in the right lower lobe, involved the lateral portion and resolved at the end of five days. With subsidence of temperature the old man arose and, despite remonstrance by the nurse, staid up all day. The next morning his right lower lobe, somewhat more anteriorly, again showed signs of invasion and temperature was up, about 102° F. A third involvement occurred in this same lung and then the process jumped to the left lower lobe and here exhibited two migrations. With each resolution the fever remitted and my hope of a favorable termination was raised only to be again dashed to the ground. In this manner the disease dragged through twenty-one days, during the last three of which the patient was mildly delirious. The case became complicated by otitis media, thick pus being freely discharged.

The prognosis in these migratory cases is serious, not necessarily on account of the intensity of the symptoms but because of the liability to failure of resistance through the protracted strain on the vital powers. Fraenkel expresses the view that the reason the mortality is not greater in this class of cases lies in the fact that the limitation of the pneumonic infiltration does not so seriously embarrass the heart and respiratory centers. "On the whole, Homburger regards its prognosis as favorable" (W. Fox).

Relapsing Pneumonia.—A distinction is to be made between this form and the one just considered. A relapse may be said to occur when, after a variable interval of time, an area which is undergoing resolution again becomes the seat of inflammation. The process, as shown by one of Fraenkel's cases, may also invade another area, but it must recur in the original zone if it is to constitute a relapse. Otherwise it might be a migration merely.

In this sense relapsing pneumonia is rare. Fraenkel states that Wagner saw but two instances out of 1,100 cases, Ruge two out of 440 cases and altogether but nine instances in the literature, while he himself observed seven instances out of approximately 1,000 cases of pneumonia. I have seen one instance, of which more anon.

The length of the interval between attacks varies within rather wide limits, having ranged between one and twenty-six days in Fraenkel's seven cases. The *duration of the second invasion* is likewise variable. In Ruge's collected instances it averaged less than five days, while in Fraenkel's it varied from two to twelve days, being three, ten, five, twelve, two, three, five, respectively.

In other regards there is nothing peculiar or worthy of special note in this type of pneumonia. All writers who make mention of it, Germain Sée, as well as those already cited, agree in the requirement that fresh evidence of the pneumonic infiltration must appear before previous signs in the same region have entirely passed away. In conformity therewith was a case observed by me in January, 1904, in consultation in Evanston, Ill.

An elderly gentleman confined to his bed with a broken leg was seized with excruciating pain in the right side of the chest on Sunday evening. The next morning his physician, Dr. Bragdon, was able to make out acute croupous pneumonia in the lower portion of the right inferior lobe. I saw the patient late that same evening and confirmed the doctor's findings.

On the third day of the disease the temperature fell to normal and the case appeared to be doing so well that my visits were discontinued. On the following Sunday, however, intense pain again recurred in that side and I was summoned to his bedside. His temperature which had been normal for three days was again elevated to the vicinity of 102° F. and I had no difficulty in recognizing fresh signs of pneumonia in the same area as that involved at first. This second attack was also short lived, about two days, and the gentleman ultimately made an excellent recovery.

Such cases are most interesting since they point to the possibility of a retention of the causative agent in a part of a lung in which its action had ceased. Prognosis is affected by the duration rather than the intensity of the affection. Such cases exemplify the uncertainty attending pneumonia and the truth of the remark previously made, that a pneumonia patient cannot be considered out of danger so long as any signs of disease persist.

Abortive Pneumonia (*Larval pneumonia*).—These are names bestowed upon mild forms of the disease whose duration does not exceed one or, at the most, two days. For this reason many clinicians have doubted the correctness of the diagnosis. I frankly confess to the same skepticism, probably because I have never had the good fortune to observe such cases. It must be admitted, however, that there are too many reports by competent observers to justify such skepticism. For instance there has quite recently come to my notice a case of a young man with aortic insufficiency which presented an initial chill, bloody expectoration, dullness, bronchial breathing, crepitant râles and elevation of temperature, in short, the signs of pneumonia. Within twenty-four hours the local signs disappeared and the temperature fell to normal.

Were it not for the character of the sputa this case would be very suggestive of the acute pulmonary congestion which Woillez depicted under the title of *Hæmopneumonia* and which French writers term *Maladie de Woillez.* When, however, one reads of similar cases reported by German clinicians, in particular Weil and Ruge, and finds that such experienced authors as Aufrecht and Fraenkel credit the reliability of their reports, he is compelled to accept abortive pneumonias as among the possibilities.

The most characteristic instance seems to be the one reported by Weil. This reporter had the rare good fortune to observe his case from start to finish since it befell a patient already an inmate of the hospital. The illness ran its course with all the characters belonging to pneumonia, except that eight days subsequently crepitant râles were still audible over the affected area (Aufrecht).

It would seem hypercritical, therefore, longer to doubt such occurrences. Nevertheless care must be exercised not to regard as larval pneumonia all cases in which only some of the symptoms are present. Thus, in the *maladie de Woillez* the expectoration is said to be fluid and sero-mucous but not so tenacious as that of pneumonia. So that although Carrière obtained pneumococci as well as streptococci and staphylococci by puncture along the margin of the dull area, Fraenkel is himself in doubt whether to regard such cases as instances of acute pulmonary hyperæmia or of mild pneumonia caused by diplococci of unusually low virulence. In like manner must be distinguished such cases as those reported by Kuehn in the prison at Mohringen, and briefly mentioned by Fraenkel as examples of febris herpetica.

In these, which might be thought to fall under the head of what Wilson Fox describes as *Latent* pneumonia, the disease began with a chill and displayed the ordinary characters of pneumonia, even to the elevation of temperature, critical defervescence and herpes, but without any evidence of consolidation upon the most careful examination. It is such short-lived and enigmatical cases that cast doubt on the efficacy of the various plans of treatment reputed capable of aborting pneumonia.

Central Pneumonia.—This term is employed to designate cases in which the exudate is so deeply seated as to escape detection by physical examination. In typical cases of fibrinous pneumonia the inflammatory process spreads gradually from the point at which dullness first appears until, at the end of the third day, the whole lobe has become involved. In the class of cases now considered the infiltration is either too limited and centrally located or, being of greater extent, is yet too far beneath the surface to give rise to physical signs at any time.

Central pneumonia is not common or at least is not frequently diagnosed. Its recognition must depend upon the character of the sputa, the temperature and pulse-respiration ratio. Rusty expectoration in the course of an acutely developed febrile affection would speak for this form of pneumonia in the absence of physical signs, and should herpes appear and the respirations be accelerated out of normal relation to the pulse, the diagnosis would be justified.

Daily examination of the chest should be made, since it is very possible for the infiltration, deeply situated at first, to spread until it reaches the periphery at some point, and so becomes recognizable. Aside from the lack of demonstrable physical findings, there is nothing in these cases to distinguish them from typical ones or to modify prognosis or treatment. I cannot now recollect having seen a characteristic example of this kind although instances have been numerous enough in which it necessitated long and careful search before the location of the small exudate was discovered.

Massive Pneumonia.—This is a term applied to rather infrequent instances of such extensive exudate as to block not only the bronchial tubes but the air-cells as well. In this condition of things the small amount of residual air not driven out by the inflammatory product is absorbed and the entire lobe, or possibly the whole lung, becomes airless. The result is that examination at the bedside discloses flatness with intense sense of resistance but no breath-sounds.

The condition closely simulates pleurisy with effusion and is very likely to lead to mistake in diagnosis. Due attention must be given, therefore, to signs of pressure on the thoracic wall and adjacent viscera since their absence would point to massive pneumonia. Should fibrinous plugs or casts be expectorated, air would again enter the tubes and bronchial breathing would become audible. This failing, resort would better be had to exploratory puncture. I have already narrated a case in which a pleuritic effusion hemmed in by old adhesions was mistaken for massive pneumonia. Displacement of contiguous organs was prevented and a differential diagnosis could have been made only by aid of a needle.

Massive pneumonias are apt to prove most grave affairs by reason of the strain on the right ventricle and the interference with oxygenation of the blood.

Latent Pneumonia.—This is a term applied to cases in which the symptoms of the lung affection are masked by those of some other complaint or are so ill defined as to escape recognition. The former is apt to be the case in children in whom the cerebral disturbances are thought to constitute the whole difficulty, and the latter in the aged when they are supposed to be simply failing from weight of years and the feebleness incident thereto. Latent pneumonia may be masked by the symptoms of delirium tremens, and hence should always be carefully sought for in drunkards. It is obvious from the foregoing that latent pneumonia is very unlikely in vigorous adults.

Typhoid Pneumonia.—This, like latent or masked pneumonia, rarely occurs in healthy young subjects, but rather in the debilitated and old. It is what is better known as asthenic pneumonia of Stokes. The phrase does not signify a conjunction of typhoid fever and pneumonia, but an adynamic type of the latter. The term is objectionable.

Bilious Pneumonia.—Much has been written about this, particularly on the continent of Europe where it would seem to prevail rather more frequently in the Summer months (W. Fox). It is an asthenic type with gastro-intestinal disturbance and icterus although the latter does not appear to be necessary to this type. It is said to occur in epidemics.

Asthenic Pneumonia.—This type of the disease is characterized by great prostration and other symptoms of intense infection presently to be described. It was divided by Leichtenstern into primary and secondary and most writers adopt his classification, in spirit at least if not in terms, since they admit the occurrence of asthenic pneumonia, both independent of and in connection with chronic disease or favoring individual conditions.

As belonging to the primary form are to be reckoned most of the cases that have manifested an epidemic tendency, i. e., that have appeared to be spread from one individual to another by infection. Thus Kühn and Kerschensteiner have each reported examples of this type among the inmates of prisons, while Müller and Butry observed similar but less numerous examples in the families of poor peasants living in an unfavorable environment. Müller's five cases concerned three residents of one house and two relatives who came to visit those first stricken. Butry's occurred among the inhabitants of a small village, and of the twenty afflicted eight died.

In this category may also be placed instances of pneumonia apparently communicated to human subjects by diseased parrots and by Morange termed *Psittakosis.* Gaston, Mallenchini, Gilbert, Fournier and others have reported such cases. In the parrots the *post-mortem* findings and symptoms were of enteritis without pneumonia, whereas in the human subjects the disease was a pneumonia with pronounced typhoid character and very fatal.

The precise nature of this pneumonia is still a matter for doubt since the results of autopsy have not always been recorded or, when so, seemed to point to a lobular distribution. The ætiology is also not clear. It is uncertain whether the bacillus found by Nocard in the diseased birds was responsible for the pneumonia in the persons who had nursed and attended the pets, or whether it was the diplococcus lanceolatus, or whether, as suggested by Fraenkel, it may not have been a mixture of both these organisms. At all events the disease was clinically a well-marked example of asthenic pneumonia.

The precise nature of primary asthenic pneumonia is likewise a matter for doubt. Fraenkel is of the opinion that the autopsies recorded by Leichtenstern and Eppinger pointed to a cellular rather than a genuine fibrinous exudate, since the cut surface was too smooth and not distinctively granular. Aufrecht, on the other hand, expresses the belief that the exudate was plainly a fibrinous one. There likewise appears to be some uncertainty concerning the bacteriology of these primary cases, although preponderance of evidence is in favor of their being due to the pneumococcus.

The primary form is infrequent and occurs more often in some countries and localities than in others. Fox states that it is rare in England and the same statement may be made of the United States. It is said to have appeared rather periodically in Stockholm (Huss cited by Fox), whereas Juergensen seems to have seen numerous cases in Basle, a circumstance which Wilson Fox is inclined to associate ætiologically with the prevalence there of typhoid fever, i. e., with the unsanitary conditions favoring the latter disease.

The *secondary form* of asthenic pneumonia is encountered in debilitated subjects and persons addicted to the excessive use of alcohol. It is met with, therefore, in public hospitals and almshouses more often than in private practice. Cases that may very appropriately be classed as asthenic are not infrequent among old people and others who are the subjects of chronic disease or of some previous malady that has lowered their vitality. This type appears to have become more common during the past ten or fifteen years, since epidemics of influenza have grown more frequent, and it probably accounts in some measure for the alarming mortality of pneumonia in our large cities.

Of course it is impossible to determine from vital statistics as collected by health boards in great cities how large a percentage of cases reported as pneumonia belongs to the genuine fibrinous form, but it is probably safe to conclude that a not inconsiderable number embraces instances of catarrhal pneumonia or of influenza pneumonia, and yet, I think, any observer will admit that during the past decade cases of secondary asthenic fibrinous pneumonia have been woefully prevalent in large centers like Chicago.

The *symptoms* of *primary asthenic pneumonia* indicate either an intense infection or a greatly lessened resistance on the part of the individual. The nervous system is profoundly affected, as shown by stupor that may deepen into coma or by delirium either low and muttering or furious. The pulse is more frequent and feebler than in sthenic cases and is often distinctly dicrotic. These unfavorable characters often appear early in the course of the affection.

The pyrexia may be high or moderate but in either event shows marked irregularity, the extremes being widely divergent. The tongue is heavily coated and dry, while sordes collect on the lips and teeth. Vomiting or a tendency thereto is present, there is diarrhœa, and the abdomen is tympanitic. Albuminuria is very prevalent and bespeaks nephritis. As the disease progresses and prostration grows, tremor develops and there may even be jactitations (Fox).

Cough is very apt to be absent, a circumstance by Traube attributed to the implication of the nervous system. If sputum is ejected, it is likely to be diffluent and bloody, of what is termed a prune-juice character. Crisis may take place, but if recovery ensues it is more apt to be by lysis, the course being very protracted.

Examination of the lungs at the bedside may reveal well-marked signs of consolidation throughout the greater part or the whole of one lobe, but it is not uncommon for such signs to be indefinite or limited to small areas. It resembles in this regard a broncho-pneumonia rather than the typical croupous form. Both spleen and liver are said to be enlarged and palpable.

On *post-mortem* examination, as already intimated, the changes do not agree closely with those of fibrinous pneumonia in that the consolidation is less firm and uniform and the cut section is not so characteristically granular. There is also a marked tendency to sudden transition from the red to gray hepatization and even to abscess and gangrene. Microscopically and bacteriologically, however, the process may be shown to belong to the typical pneumococcus variety.

Secondary cases of asthenic pneumonia resemble the foregoing very closely. This is especially so in drunkards, the group of cases most often seen in large public hospitals. An initial rigor may be experienced, but when so is not apt to be pronounced. In a considerable proportion of cases the onset is insidious and without chill or pain in the side. This is especially true of cases arising in connection with delirium tremens.

More or less elevation of temperature is present but fever is not usually high and presents marked irregularity. The skin is moist, often covered with perspiration and is apt to feel moderately cool to the hand. The temperature should never fail to be recorded in cases of *mania a potu* since, as remarked by Osler, the state of the body-heat may be the only indication of pneumonia.

Prostration is profound, and trembling of the muscles is an early feature. The sensorium is clouded and there may be coma vigil or active delirium. The bowels are apt to be loose and involuntary discharges of fæces and urine are common, but vomiting is not always present. Insomnia is a very prominent symptom and contributes to the patient's danger.

Cough is insignificant or wanting and sputa, if present, are of the prune-juice variety. The heart's action is feeble as indicated by pronounced acceleration, weakness and possibly dicrotism or irregularity of the pulse. There is often a tendency to collapse, shown by cold sweat and cyanosis. Respirations are accelerated but not so conspicuously as in sthenic cases.

The prognosis is most grave since it is this class of cases that so greatly swells the mortality from pneumonia in our city hospitals. Recovery may take place by crisis but the termination when favorable is more likely by lysis. Pulmonary abscess and gangrene are relatively common in this class of cases and account for a good many deaths.

Secondary Pneumonia.—This type of the disease was referred to in considering the ætiological influence of chronic affections. It may be seen in persons who are confined for a long time to bed and are greatly enfeebled as well as in sufferers from some long-standing disease of heart or kidneys. Its distinguishing features in most instances are its insidious onset and course and the profound asthenia which it induces. On this account it may be reckoned among asthenic cases. Because of its latency it is often overlooked at the bedside and is first discovered at the autopsy. For this reason I would impress upon the young practitioner the wisdom of von Ziemssen's advice to his students never to neglect careful examination of the chest in every new or obscure case, since only in this way can unpleasant *post-mortem* surprises be avoided.

Terminal Pneumonia.—This is only another term that is applied to cases of secondary pneumonia occurring in persons greatly weakened by some chronic and incurable malady. As remarked by Osler the temperature may become somewhat elevated and the pulse and respirations be accelerated, yet the patient be so reduced as to make a thorough examination of the lungs impossible or inadvisable. For this or for other reasons the pneumonia is not recognized *intra vitam* and hence the remarkable contrast between the statistics of pneumonia in the ward and in the dissecting room. In such

cases there may be discovered consolidation of an entire lower l
one apex.

Hypostatic Pneumonia.—By this term is signified a form of inf
of low grade which develops at the base of the lung on top of an
passive engorgement or, as it is termed, hypostatic congestion. (Se
X.) When one considers the pathogenesis of this form of congestic
class of cases in which it occurs, he cannot escape the conclusion tl
called terminal pneumonia is in many instances but another term
static pneumonia. On the other hand, hypostatic cannot be h
synonymous with terminal pneumonia since it need not be of
fatal.

This form of inflammation is preceded for an indefinite time
engorgement which has received the appellation hypostatic from the
in its being mainly if not exclusively due to the force of gra
congestion is, as a matter of fact, the result of feeble heart-a
impairment of elasticity in the coats of the engorged vessels. Thi
ment is shown clinically by impairment of resonance and enfeeb
breath-sounds at the posterior bases and, when bronchial catarrh
by fine moist râles and diminution of voice-sounds and pectoral fr

On section the lung is seen to be of a dark-red color and its c
drips with a bloody serum. It is, in a word, in the state known a
tion. This condition is recognized to be favorable for the super
inflammation, or, in other words, to furnish a good soil for tl
of such bacteria as already exist in the bronchioles and alveoli
access to them.

Hence it is that what was at first a mere hypostatic conge
become a hypostatic pneumonia under the influence of pneumococ
this occurs the dependent parts of the lung which were previously l
blood but still contained air so as to float in water, become firm, a
coarsely granular, or, in other words, present the appearance of a
pneumonia. There is, however, one important point of differenc
the overlying pleura is not involved in the inflammatory process
not show a pleuritic exudate (Aufrecht). Moreover the process
or never found in the stage of gray hepatization, owing to the b
tion of the process.

The symptoms are obscure and often disguised by those of the
malady. There may be slight elevation of temperature and the
respirations may be somewhat accelerated. Cough, however, is ab
trivial as to escape notice. Expectoration is wanting or, if presen
rusty (Aufrecht).

Upon examination of the chest there is perceived slight or c
dullness at the bases of the lungs behind, which for some unkno
is apt to be more pronounced at the right. The breath-sounds
ished or feebly bronchial and accompanied by copious fine bubbli
crepitant râles. Voice-sounds are enfeebled and pectoral fremitus
diminished. In many cases the signs are so vague that were it n
slight constitutional disturbance they would not be held to indicat

more than stasis, and indeed they are very apt to be put down to hypostatic congestion without inflammation.

There is such a striking similarity in the clinical picture to some cases of senile pneumonia that one may well inquire if some of the instances of senile pneumonia are not in reality cases of hypostatic pneumonia. Doubtless, also, many a so-called terminal pneumonia is, as a matter of fact, an infection of a previously existing hypostatic congestion.

Senile Pneumonia.—Undoubtedly elderly people sometimes react strongly to the pneumonic poison and their disease runs a fairly typical course. The exudate is lobar and compact and febrile reaction is pronounced. It was the fact just stated that led to Grisolle's utterance that in no affection do the aged manifest as high temperatures as in pneumonia. Such is not invariable, however, and my experience accords with the statement generally made by writers that the tendency in pneumonia of old people is to the asthenic type of the disease, with moderate febrile reaction.

The onset is often most insidious and cases are by no means uncommon in which the invasion of the disease was not even suspected and the detection of a full-fledged pneumonia is the first intimation of its presence. In most instances the onset is declared by a feeling of weakness and *malaise*, and physical examination shows acceleration of pulse and respiration and elevation of body-temperature. Indeed, Hourmann and Déchambre, cited by Aufrecht, assert such to be the mode of commencement in half the cases. The same authors declare that the old women of the Salpétrière arise in the morning, make their beds, walk about and eat as usual, then feeling tired lie down and soon are dead. At the necropsy a more or less extensive pneumonic infiltration is discovered.

Distinct rigor especially is not apt to be present or, if rigor is experienced it is, at the most, only a feeling of slight chilliness or shivering. Pain is rather more frequent and yet is a very inconstant feature. When present it is rarely intense, being of a dull aching character. In some cases a general "aching in the bones," to use the popular expression, is admitted, yet even this may not be so severe as to occasion complaint. Indeed, as remarked by Charcot, some of these old persons manifest no symptoms to point to extensive involvement of the lungs and only the increasing feebleness and apathy bear witness to the dangerous state of weakness.

The foregoing was well illustrated by the case of a lady of eighty whom I attended some years ago. During the greater part of a day she seemed dull and inert and showed an inclination to hug the fire though she did not make special complaint of feeling cold. By evening she appeared so quiet and stupid that I was asked to see her. In addition to the above facts the patient admitted in response to query that she ached more or less all ove the body, but not particularly in the chest. On examination she was foun to have a pulse of about ninety and occasionally intermittent, while, althoug her hands and nose were cold to touch, her temperature by mouth was fraction above 102° F. She was at once put to bed and the lungs we carefully explored, and, as had been anticipated, a small patch of dullnes with bronchial respiration and crepitant râles, was discovered at one base.

Febrile reaction is an early symptom in most cases and may be considerable, 102 to 103 or even higher. Many times, however, the temperature is not much elevated averaging below 102°. According to Fraenkel, indeed, some of these old patients present an "algid form" of the disease; the extremities are cool, the skin cyanotic and the voice almost inaudible, and in this state they succumb.

In the instances of this kind which I have seen the absence of pyrexia was only apparent and not actual as proven by the repeated observation that the temperature recorded in the mouth would be normal or subnormal, whereas, taken in the rectum it would be two or more degrees above normal. For this reason I regard mouth and axillary temperatures as very misleading in old people, particularly in pneumonia, and always insist on rectal temperatures.

The need for this care was forcibly illustrated in the case of a lady of about sixty whose pneumonia of the right upper lobe was first disguised by symptoms of cardiac inadequacy, on which account I had been summoned to see her. At my visit the second day thereafter the nurse informed me that the temperature was normal and that the patient was sleeping, and perspiring freely. In the hope of finding crisis had set in I approached the bed and was at once struck by the stertorous breathing. It required but a moment to convince me that the patient was in collapse and on taking the rectal temperature it was discovered to be 102.5°. Death occurred that evening.

Variability of the cough is another feature of senile pneumonia. It is generally very trifling or actually absent, but it may appear as a frequent slight hacking, entirely ineffectual as regards expectoration. Really troublesome cough is rarely observed in the pneumonia of old people. Sputa are more likely to be absent than present, but when there is any expectoration it is scanty and not apt to be typically rusty. I have usually noted, however, that it is sticky, in this respect conforming with that of typical cases.

On the other hand, the intensity of the disease shows itself in the circulatory and nervous system. The pulse may or may not be disproportionately accelerated from the start, depending on the state of the myocardium, as it seems to me. It is very apt to be feeble and intermittent, the intermittence being independent of any demonstrable change in the heart (Fox). The rate of respirations is also increased but not always to such a degree as to show the conspicuous perversion of pulse-respiration ratio seen in young adults. Even inconspicuous acceleration of breathing is important from a diagnostic standpoint, however, and should be carefully determined in doubtful cases. Thus, with a pulse-rate of 88 to 100 the respirations may number 28, 30 or 32 to the minute.

Early and pronounced disturbance on the part of the nervous system is very common. This may show itself as stupor that may deepen into coma, or there may be delirium. The latter is usually of the low muttering type which is most marked at night, and, with the profound prostration out of proportion to pyrexia and pulmonary findings, gives the case its asthenic character.

Why the old display such a lack of initial chill and pain, as well as of cough and sputum, and present such a distinct tendency to disorders of the brain and nervous system is hard to say. It would scarcely seem due to lessened intensity of the toxins since the patient so frequently yields to the toxæmia. Aufrecht is inclined to attribute the absence of certain ordinary symptoms to enfeebled circulation within the brain or to lessened reaction on the side of the vascular system. To my mind the lack of chill, pain, cough and expectoration is connected with that impairment of sensibility seen very commonly in aged persons even in health.

It is the physical expression of that blunting of the emotional faculties which Oliver Wendell Holmes so prettily expressed when, speaking of the gradual approach of old age, he said it is "strewed with illusions, and all its little griefs are soothed by natural sedatives." This gradual dulling of sensation depends probably upon structural changes in the cerebral cortex resulting from vascular degeneration, and, when the function of brain and nerve centers is still further depressed by acute toxæmia or possibly benumbed by carbonic-acid intoxication, it is not strange if the senile pneumonic experiences a minimum of distress from certain symptoms or if his mental faculties are clouded or perverted.

Disturbances of the digestive and urinary organs are common. There is very apt to be repugnance to food, and nausea is a frequent complaint, but I have not often seen vomiting unless occasioned by irritating or unpleasant remedies. Meteorism or diarrhœa may be present but these are not more frequent in this than in other forms of pneumonia. On the other hand, albuminuria is generally present in varying degrees, and casts are very likely to abound. Such is not strange, however, when one considers the prevalence of chronic nephritis in old people. The detection of albumin and casts is none the less serious on this account.

Upon examination of the lungs one is often surprised by the paucity of the findings. Instead of a compact consolidation extending throughout the greater part of a lobe the physician perceives tympanitic resonance with ill-defined areas of dullness; increased resistance and numerous sibilant and crackling râles, nearly or quite obscuring the breath-sounds; or at one base, perhaps, he finds a small patch of dullness with bronchial breathing and a few crepitant râles. This is all, and even this patch is partially obscured by the surrounding, somewhat tympanitic resonance. If it were not for the accompanying constitutional symptoms, he would be in doubt as to the nature of the case.

Occasionally the signs of lobar involvement are pronounced and readily discoverable, and it is worthy of note that in the aged these signs are not infrequently at the apex. I vividly recall such an instance in an old lady of over eighty. The left upper lobe presented marked dullness with tubular breath-sounds, a condition of things which, together with inconspicuous constitutional disturbance, had led a homœopathic practitioner to pronounce it a case of pulmonary infarct. It was because of this diagnosis and of the feeble heart that I was called to see the case. There was no difficulty at all in recognizing the pneumonia which, indeed, was suspected from the history, even before the patient was seen, and which terminated fatally within a few days thereafter. In this case the extent of consolidation was at first out of proportion to the degree of constitutional disturbance.

Senile pneumonia may terminate in crisis like any other, but, owing to the mildness of the febrile reaction, the critical defervescence may be overlooked. Termination by lysis is also frequent when a low grade of pyrexia may persist for several days or for a week.

Pneumonia in Infancy and Early Childhood.—Primary croupous pneumonia is undoubtedly a more frequent occurrence under the tenth year of age than is generally supposed. The reasons for this are to be found in (1) the special liability of young children to broncho-pneumonia for which cases of genuine fibrinous pneumonia may be mistaken, and (2) the likelihood of the disease to masquerade under symptoms strongly suggestive of other acute affections prevalent in the early years of life.

The following figures taken from Wilson Fox indicate clearly the frequency of the affection and its mortality: Of a total of 1,239 cases (Schroeder) of acute pneumonia fifty-six fell under one year of age, 345 between one and five years and 216 between five and ten years. Of 502 cases collected by Keller between one and eighty years of age, 295 occurred under the age of ten years. Of 898 total cases collected by Lebert two were under one year, thirty-nine between one and five and thirty-two between five and ten years of age. In other words, of 2,137 cases of pneumonia at all ages taken from Schroeder and Lebert combined fifty-eight occurred during the first year of life, 384 between the first and fifth years, and 248 between the fifth and tenth years, or 690 below the age of ten.

If we include Keller's 502 cases at all ages we have a total of 2,639 at all ages and of these 985 occurred before the tenth year, a percentage of 37.2.

Of these 985 cases in early childhood only sixty-seven died, a mortality of 6.8 per cent which, when compared with that usually given for acute primary pneumonia of adults, is reassuringly low. It is thus seen that although the disease is so common as to constitute 37.2 per cent of the cases given above, still so few proved fatal that its mortality sinks into insignificance. In a word, with exception of infants under one year of age, during which twelve months this as well as every other acute infectious disease is relatively very fatal, the mortality from fibrinous pneumonia is shown to be the least between the ages of five and fifteen years, being nearly twice as high between one and five as between five and ten.

The comparative frequency of lobar and broncho-pneumonia is shown by Holt's figures. Of 370 cases of acute primary pneumonia occurring below the age of five, 109 were lobar while 261 were instances of broncho-pneumonia. During the first six months of life seventy-three were broncho-pneumonic and but eleven were lobar, while from the sixth to the twelfth month ninety-five were broncho-pneumonic and twenty-nine were lobar. In other words, below the age of twelve months 169, or 80 per cent, were broncho-pneumonic and forty, or 20 per cent, were lobar. It is thus seen that as the child advances in age his liability to lobar pneumonia, which is the relatively more favorable form, increases and that to broncho-pneumonia decreases, although up to the age of two years the proportion of the latter to the former is still 75 to 25.

It should be stated in this connection that the great difference in the mortality of these two forms of pneumonia lies not in the ætiology or rather bacteriology of the affections, since, as is now well known and is emphasized by Holt, the diplococcus lanceolatus is capable of setting up a true broncho-pneumonia undistinguishable in its clinical manifestations from that due to streptococci. The difference is to be found in the pathology of the two forms of lung inflammation, the one being fibrinous and not inclined to impair permanently the integrity of the parenchyma, while the other is cellular and manifests a marked tendency to involve the walls of the alveoli and bronchioles in the inflammatory process. The one resolves quickly, leaving the lung practically intact, whereas the other subsides slowly and leaves more or less damage behind.

Primary fibrinous pneumonia in children may affect the whole or only a part of a lobe, in the one instance being distinctively lobar in distribution, and in the other manifesting itself as what appears to be a lobular type, and in this way simulating a patch of broncho-pneumonia. The various lobes that may be affected are given in Holt's table: first in frequency, the left lower (263 out of 950 cases below the age of fourteen) second, the right upper (176 cases) and third, the right lower lobe (168). Nevertheless, of the total number of cases 433 befell the right and 394 the left lung, while in 123 cases both lungs were involved. There is, therefore, the same tendency as in older persons for the disease to attack the right lung more often than the left. Holt's figures also show a remarkable tendency to localization of the process in the upper lobes, such being the situation in 269 out of the 950 cases analyzed.

Clinically, this disease in children displays some quite striking deviations from its features in healthy adults. Its onset is sudden and in infants is often declared by drowsiness or "dopeyness" to quote Northrop's expression. A child that but a few hours before was playing about and in perfect health becomes dull and listless and is found to have fever of 104° or 105° F. Examined physically nothing may be discovered save accelerated breathing, and in this state it may remain for several days before signs pointing to pneumonia are detected.

In some instances the invasion may be with vomiting and diarrhœa, and the illness may be thought to be gastro-intestinal. Thus I recall a female child of about twenty months who had been treated by the family physician on this supposition. When, however, signs of bronchitis appeared and I was summoned in consultation there was a small patch of dullness and bronchial breathing beneath the right scapula which, with loss of normal pulse-respiration rate and high temperature, showed the case to be one of acute pneumonia. A good recovery was made about a week later.

In still other instances the disease is ushered in by convulsions, and on this account or because of the stupor is taken to be a meningitis. Many a case, therefore, in which credit is assumed for cure of meningitis is in reality one of acute primary pneumonia. Older children may complain of headache, chilliness and pain in chest or abdomen, but if they are present in infants they are overlooked in the presence of other more apparent symptoms and because these little sufferers are unable to describe their symptoms.

Pain may not be a marked feature or, if present, may only be shown by fretfulness or crying especially when the infant is handled. It should not be forgotten that in older children the pain may not be experienced in the chest but in the epigastrium or right iliac fossa when it may give rise to a diagnosis of appendicitis. Such is particularly likely if distinctive pulmonary signs are not discoverable. If in the epigastric region and associated with vomiting, the pain may be thought to indicate gastro-intestinal disturbance, and hence in all such cases the possibility of pneumonia should always be held in mind.

Fever is generally high, 104° to 105° F., and in some cases may be the first thing that attracts the mother's attention. The pyrexia may be continuous, but often displays a tendency to irregularity or remissions. This is said to be the case especially after the first three or four days. I have known the temperature to be so irregular in a child of seven years as to create a strong suspicion of pus somewhere in the lung. The fever may decline by crisis, as in the adult, but is said to manifest a rather marked tendency to lysis.

Respirations are always accelerated and may be irregular, showing remarkable and sudden increase in rate as soon as the child is disturbed. This rapidity of breathing is the young child's manifestation of dyspnœa, and in some instances is so apparent as to be the first feature to attract the notice of the doctor as he sees the case for the first time. Respirations of 40, 50 and even 60 or 80 may be observed. So constant is such increased frequency of the breathing that in any case with early symptoms suggestive of cerebral or gastric disorders the rate of respiratory movements is likely to prove of valuable diagnostic aid. Indeed, Northrup declares that loss of pulse-respiration ratio is one of the earliest and most constant symptoms of pneumonia in young children.

The *pulse* also is accelerated, perhaps rather more so, relatively speaking, than in adults. It may run from 110 to 120 in favorable cases and in unpromising ones still higher, but, as compared with the frequency of respirations, it is sufficiently slow to present the usual inversion of pulse-respiration ratio upon which so much has already been said. *Cyanosis* may also be seen in children, and it possesses the same significance as in adults.

Cough and *expectoration* are variable, according to the age of the child. The former is rarely absent even in infants, but is not attended with the expulsion of sputa, because of the well-known habit of these young patients of swallowing their expectoration. That it is rusty the same as in adults is attested by von Ziemssen's observations, who is said to have seen such characteristic material mixed with the matters vomited. Older children are sometimes greatly plagued by cough and then are quite likely to raise rusty, viscid sputum.

Anorexia, restlessness, insomnia, prostration, etc., are not peculiar to the pneumonia of childhood and depend in their intensity upon the severity of the infection, duration of the disease, and resistance on the part of the patient, the same as in persons of older age.

Convulsions are among the symptoms which are liable to occur in infants, and these are justly alarming because of their gravity. They may come on suddenly and unexpectedly toward the time when crisis should be looked for, i. e., late in the course of the disease. At this time they are very apt to be fatal, having proved rapidly so in a case seen by me in the Spring of 1904. According to Holt they may or may not indicate the occurrence of meningitis as a complication. They were dependent upon meningitis in two out of three instances observed by Holt, as he says in the second edition of his work.

The great liability of fibrinous pneumonia in young children to be complicated or associated with involvement of the pleura should be especially mentioned. It would seem as if pneumococcus empyema were relatively more frequent and extensive in children than in adults. Interlobar empyema is doubtless present far more often than is suspected or detected owing to the great difficulty of its diagnosis in many cases. Details of such an instance will be found in the chapter on Pleurisy, under Interlobar Empyema. It should be borne in mind in the recognition of empyema that, if extensive, it may be pent up under conditions of such high pressure as to permit and even facilitate the transmission of breath-sounds of a distinct bronchial character. If in any given case of fibrinous pneumonia in a child the fever persists for an unusual length of time after pulmonary signs have abated or greatly moderated, it should create a suspicion of empyema and lead to careful search for the same.

The *diagnosis* of primary pneumonia in young children is often a matter of great difficulty because of the liability of the symptoms to simulate those of other complaints and of the tardiness in development of distinctive pulmonary signs. The points to be relied on in the beginning of the illness are (1) the suddenness of onset, (2) "dopeyness" or somnolence, (3) high fever, (4) the loss of normal pulse-respiration rate, this last being invariable, although not always pronounced, and requiring for its detection repeated careful counting of pulse and respirations.

The pneumonic patch is often very slow in reaching the surface, three to four days, and even then may be so small that minute care is essential in examining the chest. *Percussion* may fail of its detection because too strongly performed, as well as on account of the fact that percussion-strokes over the lower portion of the chest may set up vibrations within the air-containing viscera of the abdomen and thus produce a tympanitic resonance which obscures whatever dullness may exist.

For this reason I rely largely on the results of *palpation* of a young child's chest. If the outspread hands are made to grasp the two halves of the thorax simultaneously from behind (see Fig. 27), and the infant then be induced to cry or moan, the vocal vibrations are readily appreciated, even when the zone of consolidation is limited. Of course this means of investigation does not invariably yield definite results. The area may be wholly centric, or the baby may be too drowsy to cry or even to groan when lifted up out of its comfortable position; but if palpation after this method is systematically practiced it will afford valuable information in very many instances.

Auscultation is, in most cases, the most valuable method of eliciting pulmonary signs. These are bronchial breathing either with or without fine crackling râles over a circumscribed area on the back or in the axillary region on one side. Soon or late such a patch is pretty sure to develop and hence the physician must carefully go over the child's chest every time his visit is made until he detects what he is in search of.

Important information may likewise be derived from a count of the white blood-cells and by an estimation of the chlorides when urine can be obtained. Given the symptoms mentioned above, a leucocytosis of 18,000 to 20,000 or more, diminution of chlorides, and in particular a pulse-respiration rate of 1 to 3½ or 3, still better 1 to 2, and the diagnosis of pneumonia may quite confidently be made even without characteristic pulmonary findings.

Lastly, a few words concerning the *prognosis* in primary fibrinous pneumonia in children. The table already given shows a death rate of 30 per cent below the fifth year. This disease is undoubtedly a serious one in young children, especially under the age of three years, and yet I am persuaded that the table referred to places the mortality too high for simple uncomplicated cases seen in children of the better class. In foundling asylums and children's hospitals the mortality may be relatively high, but in a child with good environment and proper medical attention, good nursing and a previously healthy constitution, the prospect of recovery is extremely good.

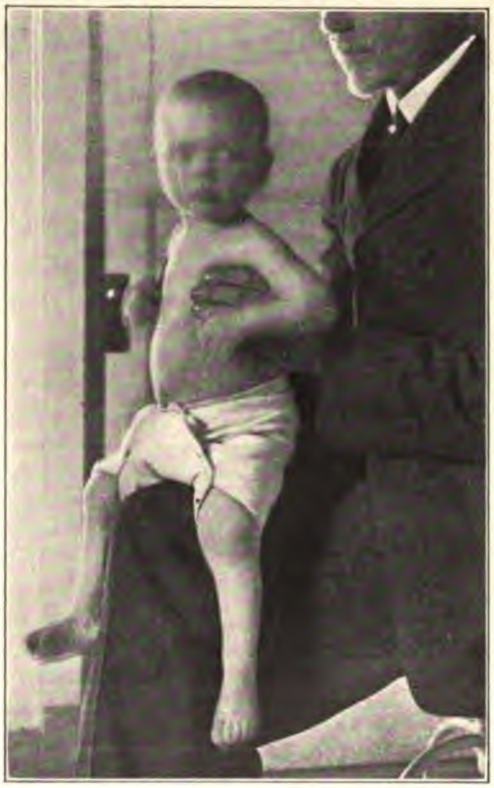


FIG. 27.—Method of palpation of the infant chest.

Prevalence and Mortality.—Few diseases of modern times are more to be dreaded, and justly so, than acute croupous pneumonia. This fear is owing to the increasing prevalence of the disease, to its high mortality and to the prominence given in the public press to its frequency and danger. In some of the large cities of the United States the malady has raged with such fury in recent years and has struck down so many prominent persons in the midst of apparently excellent health that the approach of Winter has become a source of apprehension on the score of the liability to pneumonia during that season.

The general opinion that this affection is steadily increasing or at least has increased during the past ten or fifteen years seems borne out by the figures presented by E. F. Wells in a paper read in June, 1904. According to these the annual death rate in New York, which was 1.3 per thousand of population in the decade of 1804 to 1813, rose to about 2.5 per thousand in the last decade of the century. In Philadelphia the death rate of 1.2 per thousand between the years 1861 and 1870 rose to about 2 per thousand for the years 1893 to 1902, while in Chicago the death rate of 0.5 in the decade 1851 to 1860 was tripled in the final decade of that century.

There may be many factors entering into this increase in the prevalence of pneumonia, such as greater virulence on the part of its bacterial cause, the influence of influenza as a predisposing factor, etc., but when the comparative favorableness of the disease among the soldiers of Germany (3.6 per cent among 42,476 cases) is contrasted with the figures soon to be given, it would seem as if the increased population in our great centers, and the unhygienic conditions attendant thereon, might be largely responsible for the greater mortality from pneumonia now than formerly. Yet, whatever be the conditions governing the mortality from this disease, or whatever be the actual facts concerning its increase, the death rate in itself is appalling and may well excite alarm in the minds of the profession and the laity alike.

Mortality tables, unless comprising very large numbers, are apt to be misleading. This is particularly true of pneumonic fever, since the tabulated cases are drawn from widely different sources, as from hospital or from private practice, and embrace patients of diverse ages besides different types of the disease, as secondary or primary, asthenic or sthenic. Hence we find a wide range in the death rate recorded by observers.

On consulting the tables collected by E. F. Wells we see that Bell, Brenchley, Frohmüller, Hegele, Hulava, Müller, Robinson, Salama, Stoahml, Wittich and Zeigeli all reported cases without a single death, whereas Hourmann and Déchambre recorded 109 cases with a mortality of seventy-six, or 70 per cent, and Becquerel twenty-eight with a mortality of twenty-three, or 82 per cent. Such wide discrepancies as these assuredly warrant the conclusion that too many diverse factors govern the death rate to justify deductions from small numbers. It is quite otherwise when very large numbers are taken as a basis of reckoning and hence we must commend the spirit shown by Wells in gathering figures from all possible sources as he has done.

In his latest published report (June, 1904) he gives tables of the mort ity of pneumonia comprising 465,400 cases with 94,826 deaths, or 20.4 p cent. Even this average cannot be taken as a basis for prognosis in es mating the chances of any single pneumonic patient, since the many tab that entered into Wells's figures were derived from all sources. Moreov when we study the figures taken from the annual reports of some sin hospital, e. g., the Wiener Allgemeines Krankenhaus, we are struck by the variations in the death rate in different years, although the assumption is fair that conditions during the various years could not have materially altered. Consequently we must now consider the numerous factors that influence the mortality. The annexed chart taken from Wells's paper shows the yearly fluctuations very beautifully, and also the claim made by the author of the chart that pneumonic fever has been steadily increasing on the whole.

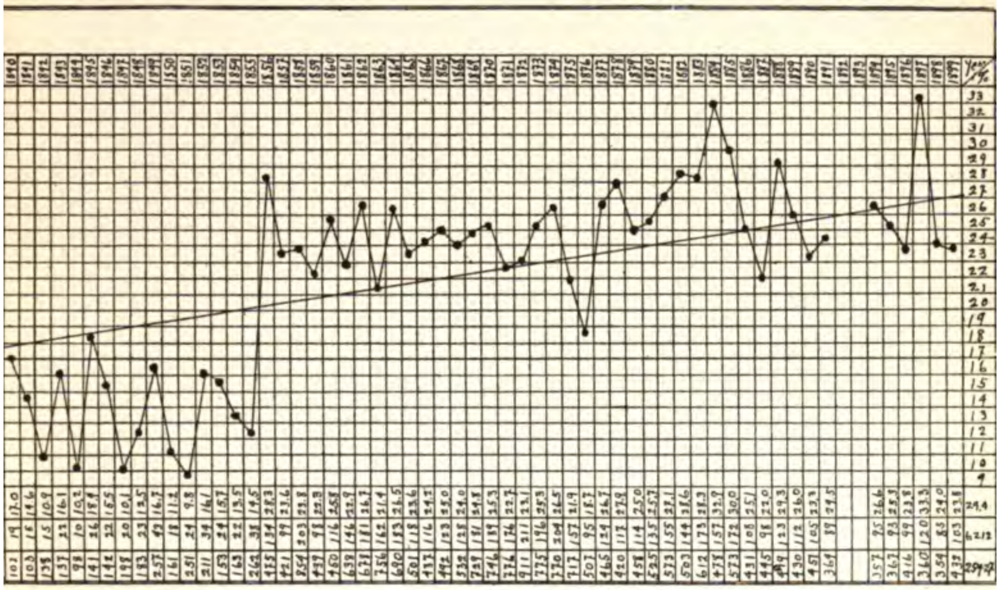


FIG. 28.—Chart showing yearly mortality from pneumonia in Vienna General Hospital (Wells).

Prognosis.—A pneumonic patient can never be called out of danger until he is well. This dictum does not apply with equal force to all forms of the disease nor to all individuals, for, paradoxical as it may appear, the sthenic form with its violent onset and stormy course is relatively more favorable than the asthenic type characterized by lower temperature and less pronounced pulmonary disturbance. Osler gives the average mortality of pneumonia as from 18 or 20 to 40 per cent, whereas Liebermeister states the mortality of the sthenic form to be from 3 to 10 per cent.

So many diverse factors influence prognosis that it is difficult to estimate their relative value. We have to reckon with the virulence of the infection on the one hand, and on the other with individual resistance and all the various elements entering into such resistance. The violence of the symptoms in the sthenic form seen in the young and healthy, and conversely, the prostration marking the asthenic type in the aged out of all proportion to the intensity of the fever and extent of lung-involvement, would seem to indicate that the prognosis in any given case depends largely upon vital resistance.

Age.—Other things being equal, the prognosis bears a direct relation to age. This is shown by the annexed table, which was taken from Aufrecht and which was copied by him from Fraenkel and Reiche. Of 1,130 cases of primary pneumonia death occurred according to ages as follows:

1– 5	years	9	times in		30	cases	= 30.0	per cent
6–10	"	2	"	"	52	"	= 3.8	"
11–20	"	11	"	"	219	"	= 5.0	"
21–30	"	37	"	"	355	"	= 8.7	"
31–40	"	57	"	"	231	"	= 24.7	"
41–50	"	53	"	"	135	"	= 39.3	"
51–60	"	28	"	"	65	"	= 43.1	"
61–70	"	15	"	"	28	"	= 53.6	"
71–80	"	13	"	"	15	"	= 86.7	"

From these figures it appears that after the fortieth year the mortality grows steadily and reaches its maximum between seventy and eighty, at which period of life the rate of death is truly alarming. In the first half decade the mortality exceeds that between thirty and forty while between the ages of five and thirty, or that period of life when the sthenic type of the disease prevails, there are 59 fatal cases out of a total of 626, or a mortality of 9.4 per cent.

Such figures show plainly the influence of age and that, other things being equal, healthy young adults have a good prospect of recovery from acute fibrinous pneumonia. On the other hand, the fatality is very great in the senile form of the disease. This is not at all surprising, however, when we consider how readily old persons who are apparently vigorous lose strength during acute illness of whatever nature.

Regarding the prognosis of croupous pneumonia in children opinions are divided. In the table of age-mortality just given it is seen that between five and ten years the death rate was extremely low both absolutely and relatively, but two out of fifty-two cases or 3.8 per cent which agrees very closely with the mortality among the German soldiers who are presumably among the most robust in the community. Below the fifth year and especially under the third the prognosis is more grave and yet, even as regards children of this tender age, opinions are at variance.

Thus, to quote from one of Wells's papers, we find that Trousseau is said never to have lost a case of genuine fibrinous pneumonia among such young patients, and von Ziemssen recorded but seven deaths out of 201 cases in infants or 3.3 per cent. Baginsky considers the danger slight, while Vogel who thinks the danger considerable under two years takes a favorable view of cases over that age. Sturges says the old are likely to die, the young t recover, and D'Espine that the danger in infants is very slight. On t other hand, Steffen reports a mortality of 13.8 per cent in children an Funcke puts it at 10.3 per cent.

Such differences can only be explained, I think, on the ground th the observations made by these various authors covered quite a wide ra of ages. The period of infancy includes the first two and a half year life, and childhood all the period from two and a half to fifteen years. Whereas the fatality is low from the first year upward and grows less as we proceed toward the third decade of life, the danger increases as we descend in the first twelve months of life. Primary croupous pneumonia is very rare in young infants, yet it does occur and, although not nearly so serious a malady as broncho-pneumonia, it cannot be regarded lightly in infants under one year, the prognosis being progressively more grave the younger and less resisting the patient. Nevertheless, I have known a baby of six months to recover from a genuine lobar pneumonia of the right lower lobe.

As respects older children it may be stated that Jürgensen lost but four out of 171 cases below the age of ten, Gerhardt but one out of forty patients ranging in age from six to sixteen, Barthez but two out of 212 between the second and fifteenth year. On the whole, therefore, the prognosis in childhood may be regarded as extremely hopeful provided the child was previously healthy.

Sex is thought to exert peculiar influence on both the liability to pneumonia and the danger from it. Females suffer relatively less often from the disease, but seem to be more liable to a fatal termination, a fact which finds its explanation in several circumstances; such as the greater susceptibility of the nervous system in women and their premature loss of vitality from childbearing (W. Fox); their greater liability to double pneumonia (Huss); the perils of abortion during pregnancy and pneumonia (Grisolle and Klautsch); the liability to uncontrollable hæmorrhage following miscarriage during pneumonia (Jürgensen); the increased intensity of pneumonia coming on during menstruation (Grisolle); and the unfavorable influence of chlorosis upon the course of pneumonia (Huss), a blood state notoriously more common in the female sex.

The relatively greater mortality among females is shown by the figures collected by Edward F. Wells, whose zeal and thoroughness in the collation of statistics are most commendable. Of a total of 23,853 cases among males, taken from fifteen different sources, there were 4,643 deaths or 19.4 per cent, whereas of 9,743 cases in women there were 2,798 deaths or 28.7 per cent. On calculating the death rate of the combined cases among both sexes it is found to be 22.1 per cent, which is less by 6.6 per cent than that among the females.

The figures just given are supported by others taken from Funcke (17 per cent and 23 per cent respectively), Lebert (16 per cent and 21.8 per cent), Bamberger (8.7 per cent and 16.6 per cent), Roth (16.2 per cent and 23.6 per cent), Munich General Hospital (16.6 per cent and 23 per cent), and Vienna's three great hospitals (20.7 per cent and 30 per cent). Such differences in the death rate between the two sexes are too striking and unanimous to permit the assumption of accident. Neither can we assume a greater virulence on the part of the germ in the female cases. The only conclusion permissible is that women are less resisting to the pneumonic poison. As regards the influence of menstruation on the pneumonia, Wells thinks it is favorable rather than otherwise. The influence of pregnancy will be considered later on.

Race.—The importance of race seems to be variously estimated by writers. Some say nothing about it, while others state in positive terms their conviction that the colored races are less resisting to pneumonia than are the Caucasian. Negroes are said to be particularly susceptible (Aufrecht) to this malady and to evince a high mortality (Wells). Personally I am inclined to the opinion that if the negro race withstands pneumonia badly, it is not due to any racial peculiarity per se but to the unfavorable environment and careless, often dissipated habits of the negro.

Locality.—Although the death rate differs more or less in different countries and cities, such differences appear to me to depend not so much on climatic peculiarities as on other conditions such as overcrowding, habits, etc. *Altitude* is thought by many to be of influence, but H. W. Hoagland, from an analysis of statistics at his command, concluded that pneumonia is not materially more fatal at an altitude of 6,000 or 7,000 feet than at the sea level.

The Degree of Fever.—Great differences are seen in the temperature of pneumonia. In young adults or individuals with good resisting power the temperature is apt to average high, while in the old and debilitated it is generally low or moderate. The latter class of cases furnishes the highest mortality and hence the degree of fever is, within certain limits, an index of the individual's power of resistance. It has a bearing, therefore, on the prognosis.

William Ewart, from an analysis of 1,000 pneumonias occurring in the London Hospital during a period of ten years, found the mortality increased proportionately as the temperature averaged higher or lower than 103° F. Accordingly he concluded that this might be looked upon as a safe point. Wilson Fox on the contrary regards an average temperature of 104° F. as furnishing the best prognosis. This opinion is corroborated by the figures given by Preble, according to which the mortality is less between 103° F. and 105° F. than between 102° F. and 103° F., being 26.35 per cent and 27.4 per cent respectively.

Strangely enough, however, Preble's table shows a mortality of 23.1 per cent between 100° F. and 102° F. which would seem to indicate this latter as the safest average temperature. Figures are in themselves misleading, however, since they do not take into consideration many other factors that influence prognosis. All authors agree in the statement that temperatures averaging above 105° F. or below 100° F. indicate danger, and that above 106° F. the mortality becomes very great (68 per cent, Preble).

Patients displaying persistent hyperpyrexia (105° F. to 107° F.) may recover under vigorous antipyretic treatment by means of cold, but the outlook is very grave. If the temperature average 107° F. or higher the outcome is probably always in death. Such high temperatures indicate great virulence on the part of the germ as well as vigorous reaction by the patient and in the struggle between these two forces the former is likely to conquer.

It is said to indicate an unfavorable prognosis if the fever is refractory to measures for its reduction, or if it rises again quickly after having been lowered, or if its course is continuously high. Remissions and even intermissions are not grave, on the other hand, so long as they are not due to complications. It is a grave omen if the surface of the body becomes covered with a cold sweat and the mouth or axillary temperature is normal or subnormal whereas that in the rectum is still elevated. This is a condition of collapse and may sometimes prove misleading. As has been previously stated, I have known a nurse to report a patient under such circumstances as better because the axillary temperature was normal, and yet by rectum it was 102.5° F.

Circulatory System.—The pulse is not so trustworthy a guide in forming a prognosis as are the heart-sounds and of these the pulmonic second. Nevertheless, marked frequency of the pulse is not seen in pneumonic patients who are successfully withstanding the pneumococcus poisons. On this account the physician should view with concern a persistent pulse-rate of 120 or higher, and if at any time a pulse previously satisfactory begins to increase in frequency and continues to do so, it is to be taken as an unfavorable sign of the action of the poisons on the myocardium.

It is likewise of evil import if the previously regular pulse begins to be irregular in force and volume, perhaps also in time. Intermittence is also of grave significance, but to my notion is not always so serious as great rapidity or the loss of regularity in size and strength of the pulse-waves, for the former may be due to some gastro-intestinal or nervous disturbance, whereas the latter is an indication of failing heart-power. The only really trustworthy guide as to the integrity of the heart-muscle is to be found in the heart-tones.

As soon as the exudate has filled a portion of the lung and has led to compression of the pulmonary capillaries in that district, blood-pressure in the pulmonic system is raised. There is then a corresponding intensification of the pulmonic second sound which may be almost painful to the ear. Under these circumstances the right ventricle is strong, somewhat dilated and, after a number of days, actually begins to take on a certain degree of hypertrophy. Therefore, so long as the second sound in the second left interspace near the sternum remains clear and ringing it may be assumed that the right ventricle is still equal to the strain.

Three factors are at work endeavoring, so to speak, to break down the integrity of the heart-walls: (1) the infection, (2) the fever, (3) the mechanical impediment to circulation through the lungs. Before the infectious nature of lobar pneumonia was understood clinicians used to consider only the effect of the two latter, but now it is well known that the toxins of acute infections exert a powerful influence for ill on the heart. This is particularly true of the disease we are now considering as shown by certain facts. It is not uncommon to see individuals go through an illness without serious indication of heart-weakness in spite of their having two or even three lobes absolutely solid, to judge from physical signs.

So also with high fever: The temperature may remain for a week at 105° F. without exerting any appreciable ill effect on the myocardium, despite the established fact that pyrexia tends to exhaustion and degeneration of muscular tissue. On the other hand, cases of acute pneumonia are, alas, only too frequently seen in which, almost from the beginning without extensive exudate and with moderate or indeed low fever, the heart displays serious and even alarming asthenia.

This is not necessarily, although possibly, an acute myocarditis, but may be a simple toxicity of the myocardium or of the nerve-centers, in particular the vaso-motor center in the spinal cord. When, therefore, the pulmonic second sound begins to grow feeble and muffled it is an indication of the gravest peril; and unless cardiac power can be sustained by appropriate treatment it portends a fatal termination of the case. It is largely because in the aged the heart-muscle is feeble through degenerative changes that senile pneumonia is so fatal, and conversely because the heart of the young is sound and capable of withstanding the influence of toxins that in them the prognosis is relatively good in croupous pneumonia.

Cyanosis is another symptom of grave danger whose appearance has always been and is justly viewed with apprehension if not with alarm. In its lighter grades it may be a manifestation of defective oxygenation or of an overburdened venous system and right heart. Intense cyanosis, on the contrary, is an expression of toxic paralysis of the vaso-motor centers in the spinal cord and consequent paresis of the capillaries. Such cyanosis is therefore an early signal of circulatory failure and if not successfully combated by vigorous cardiac stimulation is a precursor of inevitable dissolution through failure of heart power. The physician should be on the constant watch for increase of this symptom and if this be associated with weakening of the pulmonic second tone or increasing rapidity of the pulse he should institute prompt and energetic treatment to sustain the heart. Few patients recover from pneumonia after cyanosis has become well marked.

Leucocytosis is, on the whole, to be regarded as of favorable prognosis, since it evinces a strong effort on the part of the system to combat the infection. On the contrary an absence of leucocytosis throughout the course of the disease or a decrease in the number of white cells after an initial increase is generally to be looked upon as an unfavorable sign. Both of these statements are open to exceptions, however. In spite of a very marked increase in the number of white cells a patient may yet die from the intensity of the infection, and conversely an absence of leucocytosis may be due to the mildness of the disease, and hence the outlook may be good although the leucocytosis does not appear. In conclusion it may be said that pronounced leucocytosis in the presence of a severe pneumonic infection does not necessarily mean recovery; whereas absence of the same under similar conditions means the probable death of the patient.

Respiratory System.—Rapidity of breathing is not in itself necessarily a bad symptom, since it may be due to pain independent of the extent of lu involved. When, however, the respirations reach 50 or 60 or more t either point to great and dangerous diminution of lung capacity from tensive exudate or to some mechanical obstruction, as bronchitis, or to p monary œdema in parts not invaded by the exudate.

The breathing may be temporarily greatly accelerated by passing caus but if persistently much above 40 should excite apprehension and warr repeated examination of the lungs for the detection of signs of œdema or of extension of the disease. The invasion of new areas in the same lung or in the opposite lung is, as might be expected, of grave import, since it means delay of the crisis with all its attendant dangers. When the patient becomes insensible to his dyspnœa and ceases to cough it indicates loss of reflex irritability and is a grave prognostic omen.

Pulmonary œdema is of the worst possible import and, if general, is to be regarded as the beginning of the end. It is still not settled whether œdema of the lungs in acute pneumonia is to be considered as a sign of heart failure or as but another manifestation of the pneumonic infection, as Rivalta's observations appear to show. Of fifty-two fatal cases of pneumonia that came to necropsy this author found pulmonary œdema in thirty-three or 63.4 per cent. In twenty-two cases an entire lung was involved by the œdema, in six the base of one and in two the bases of both lungs, in three an upper lobe.

In the œdematous portions Rivalta was always able to detect the presence of Fraenkel's diplococcus, while the alveoli in the œdematous parts showed a beginning exudate. This was characterized by the presence of red and white blood-cells intimately blended with desquamated epithelia. Whether Rivalta's observations shall be confirmed and alter our conception of the exact nature of pulmonary œdema in this affection or not, it will not change the fact that this condition is to be regarded as of the gravest possible import and to call for renewed efforts to save the patient.

Cerebral symptoms, as delirium and coma, very naturally occasion concern. The former, coming on early in the course of pneumonia and associated with high fever, may be the result of brain-irritation from the pyrexia or toxins, but is apt to render the physician apprehensive lest it be the precursor of meningitis. Developing late, delirium is due to exhaustion and should excite concern on this account. Appearing after the crisis it is less serious since it will be likely to subside under appropriate stimulating treatment.

A comatose state, if associated with scantiness of urine, is grave and very naturally excites alarm inasmuch as it involves the possibility of uræmia. Connected with restlessness, *subsultus tendinum* and picking at the bedclothes, cyanosis and feebleness of the pulmonic second sound, it portends a not very distant termination in death. The opposite state of persistent wakefulness also adds to the gravity of prognosis. The insomnia is an expression of the infection and as such serious, but it also robs the patient of much-needed rest.

Convulsions are always alarming and yet their significance from a prognostic standpoint depends largely on the date of their occurrence. In young children they not infrequently usher in the pneumonia and then are of diagnostic rather than prognostic significance. When, on the other hand, convulsions appear late in the malady they are apt to indicate meningitis, but whether so or not they are likely to usher in a fatal termination, often very speedily.

In adults convulsions are unusual and yet that they do sometimes occur was proven by a case that has been referred to previously. A man was admitted to Ward 4 of Cook County Hospital in a state of unconsciousness and directly thereafter began to display convulsions that were typically epileptiform. The right half of the body was the more strongly convulsed. The spasms recurred at short intervals and necessitated the administration of sedatives. A subsequent examination of the chest disclosed unmistakable signs of pneumonia from which the man died the following day. The autopsy revealed the pneumonia and in addition an acute meningitis.

Extent of Lung Involved.—Prognosis is also affected by the extent of lung involved in the pneumonic process, since the more extensive the consolidation, the greater is the respiratory embarrassment and the greater the strain put upon the right ventricle. Double pneumonias therefore are generally regarded with apprehension, although they are by no means incompatible with recovery. In such cases the fever is apt to be high which is not only of itself less favorable, but also speaks for an intense infection.

Seat of the Pneumonia.—The situation of the process as it influences prognosis is a matter of uncertainty. Regarding the relative danger of right and left side pneumonias statistics are not very reliable.

The figures given by Aufrecht make pneumonias of the right lung, which as was to be expected outnumber those of the left, somewhat more fatal, the mortality being 15.3 and 12.8 per cent respectively, a difference too slight to be of much weight in estimating prognosis.

Apex pneumonias are quite generally held to be more dangerous than those of the base. This was the teaching of Andral and Grisolle, but was denied by Briquet and is shown by statistics to be scarcely the case. Wilson Fox is of the opinion that the apparently greater mortality of apex pneumonia is to be attributed, not to the fact of the situation *per se*, but to its more frequent occurrence in the aged, in drunkards and in debilitated subjects, and to its likelihood of being tuberculous.

Complications.—The danger in pneumonia is enhanced by complications and some of them, as meningitis and pericarditis, render the prospect of recovery very remote. The former, indeed, may speedily prove fatal. Extensive bronchitis affecting the other as well as the pneumonic lung adds to the patient's peril apart from the fact that it denotes widespread inflammation and therefore furnishes a grave prognosis.

Acute endocarditis is most serious since it is apt to prove malignant and to cause death some time after the pneumonia has resolved. An endocarditis may, however, be outlived though likely to leave a valvular lesion behind. Pleurisy of the opposite side adds to the gravity of the pneumonia, especially if the exudate be purulent but does not in itself prevent recovery. Arthritis otitis media, parotitis, etc., are serious, yet need not unfavorably affect prospect of life, although they do materially retard convalescence.

Pulmonary abscess may be recovered from without surgical interver but gangrene, unless operable, is likely to prove fatal. Both add greatl the gravity of prognosis. Septic diarrhœa is also a serious complica since it betokens intense infection and contributes powerfully to exhausti Acute nephritis adds an element of danger but does not preclude recove

It is less serious than the acute glomerular inflammation observed in the course of streptococcus infection.

Chronic diseases also constitute serious complications in acute pneumonia. This is particularly true of cardiac lesions, but aortic valve diseases are less dangerous than mitral affections since they have impaired the integrity of the left rather than of the right ventricle. The mechanical impediment to pulmonary circulation occasioned by the pneumonic exudate adds enormously to the strain to which the right heart is already subjected by reason of a mitral defect, and hence cardiopaths with mitral disease, especially stenosis, endure pneumonia very badly.

Chronic myocarditis is also a grave complication since a degenerated heart-muscle is likely to succumb easily to the depressing effect of pneumotoxins, and yet one is sometimes surprised at the resistance shown by such hearts. The inference is warrantable that, after all, the ability of the heart to withstand pneumonia depends mainly on the intensity of the infection.

Chronic nephritis and hepatic cirrhosis are complications that furnish a very unfavorable prognosis (Aufrecht). In fact, this author states that they almost always exert malign influence over the course of a pneumonia. That chronic Bright's disease should endanger life is readily comprehensible when we consider that such kidneys are particularly liable to acute inflammation in the course of infectious maladies. At the same time their eliminative power is lessened, and as the prognosis is largely determined by the ability of the kidneys to eliminate, it is clear that a chronic nephritic who is doing well so far as his lungs are concerned may yet be unable to withstand the toxæmia.

Pregnancy.—The prognostic relationship existing between pregnancy and pneumonia concerns not so much the termination of the latter as of the former. Other things being equal a pregnant woman may survive an acute pneumonia as well as a nonpregnant woman and yet that the danger is enhanced by pregnancy is quite generally recognized. The danger comes, however, through the premature delivery of the fœtus which statistics prove to be the chief source of peril. In other words, a pneumonia is very apt to occasion abortion or miscarriage, on the third or fourth day of the disease (Klautsch).

Fraenkel is of the opinion that in such premature expulsion of the fœtus lies a positive source of danger to the already overburdened right ventricle. In proof thereof he cites Georgii's figures which showed that of twenty-eight cases of pneumonia in which abortion was occasioned *prior* to the seventh month death took place in nine. Of forty cases in which premature birth occurred *after* the seventh month, death was the result in nineteen.

Chronic Alcoholism.—In no class of individuals is the mortality from pneumonia so high as among hard drinkers. This is attributable to the low resisting power of such persons and to the speedy development of certain symptoms that tend to rob the patient of what strength he may possess. These are delirium and insomnia. This combination is serious in any case of pneumonia and when, as is so often seen in the pneumonia of drunkards, the condition of coma vigil develops, a fatal prognosis may generally be given.

The death rate in this class of cases is put down as from 48 per cent to

50 per cent, and even higher. One is often surprised by the suddenness with which a robust-looking man who admits the abuse of alcohol will develop grave symptoms and die from pneumonia which judged by the extent of lung involved is mild. The high death rate of 47 per cent recorded among the pneumonic patients admitted to Cook County Hospital in 1903 is attributable in great measure to the large percentage of alcoholic cases.

Delay of Crisis.—Notwithstanding the apparent fact that crisis is more likely to occur after than before the fifth day, still the longer its delay beyond the fifth day, the less favorable becomes the prognosis. Not only is the sufferer's strength more sorely taxed, but there is greater liability to involvement of the nerve centers and to failure of the heart-muscle.

Moreover, Ewart's statistics previously cited appear to show that the crisis is in itself not devoid of peril. The heart frequently flags seriously at this time, as evinced by feebleness and intermittence, a fact which Ewert attributed to the sudden withdrawal of the spurlike action of the pyrexia. Whether this be the correct explanation or not, the physician, while yet eagerly hoping for the crisis, should not be unmindful of the circumstance that many a patient has succumbed after defervescence has taken place.

In conclusion it should be recognized that in some years all pneumonias are intense and display a far larger percentage of mortality than at other times with precisely the same therapeutic management. This is a matter of undeniable clinical observation the same as is the striking difference in frequency in some years as compared with others. This evident intensity or, as may be said, malignancy of the disease, in some years and at some places the disease being so severe and so frequent as to merit the term of an epidemic, is probably to be explained on the hypothesis of greater virulence of the pneumococcus. If such is not the explanation then there are at work some accessory factors which have as yet escaped discovery.

It seems to be a matter of quite general observation that since the advent of influenza and its almost endemic character the mortality from pneumonia has become increased. It might seem at first sight as if this were due to a mixed infection. It is instead because a pneumonia occurring in an influenza patient is secondary and attacks one already enfeebled by *la grippe*.

Finally, I desire to mention a prognostic sign to which Dr. E. F. Wells, an unusually keen observer, has called my attention. He says, "During the pneumonic attack reflex sensations are weakened in proportion to the danger. Now, a dyspnœa, which is readily recognized by the observer, but which is not appreciated by the patient, is of grave augury, and when accompanied by a rising and falling of the trachea and an unobtrusive clicking noise with respiration, which persists after coughing, the patient has, in my experience, invariably died. The prognostic value of this syndrome lies in the fact tha it antedates all others which certainly denote the approach of death. On the other hand, when, late in the attack, the patient wakens from a sound sleep and sneezes, his safety may be assured."

On one occasion I had an opportunity to verify Dr. Wells's statements a regards the significance of this click. On January 1, 1903, I detected thi peculiar sharp inspiratory click in an aged lady with pneumonia and as sh was apparently sinking I predicted the near end. She rallied to our surprise, however, in a most remarkable manner and it looked as if she would belie my prognostications. Yet, after thus reviving for a day she ultimately died, without my having again heard this ominous click with its original distinctness.

Causes of Death.—The immediate cause of death in the overwhelming majority of cases of acute pneumonia is paralysis of the heart. Prior to the establishment of the infectious nature of this disease heart-failure was attributed to the exhausting effect of high fever aided by the mechanical distention of the right ventricle. Although such may be contributing factors, the real cause of cardiac asthenia is the toxæmia. This may affect the myocardium directly, as held by Fraenkel, or indirectly in consequence of vaso-motor paresis.

Dissolution is generally preceded by signs of heart-weakness, such as increasing frequency of the pulse, but now and then instances are observed of instantaneous arrest of the heart when the patient appeared to be doing well. In the Winter of 1905, in connection with Dr. F. W. Mercer, I was attending an elderly gentleman who died in this manner. His pneumonia was of the sthenic type and primary. On the morning of the sixth day his pulse was 108 and his condition appeared hopeful when, without warning and immediately after having taken some liquid from the nurse, he fell back on the pillow and expired.

Death under such circumstances is attributed by some to heart-clot, an opinion that seems supported by the *post-mortem* discovery of thrombi in the cardiac chambers. So far as I can determine from perusal of recent literature such clots are thought to be *post mortem*, not *ante mortem*, and the sudden arrest of the heart's action is considered due to diastolic arrest, the result of a toxic paralysis of the myocardium.

Another not infrequent cause of death is pulmonary œdema, either general or limited to portions of the lungs free from the exudate. Whether a result of mechanical congestion from failing heart-power or a manifestation of pneumococcus action on alveoli previously unaffected (Rivalta), the œdema seems to become the immediate cause of dissolution in a considerable percentage of cases which until its sudden advent appeared to be doing well.

The postcritical collapse occasionally observed as a cause of death has been attributed by Bolinger to a veritable oligæmia produced by the withdrawal from the circulation of the large amount of blood contained in the exudate. This, he claims, acts in the same manner as a severe hæmorrhage. Sufficient blood is not supplied to the heart for its nutrition and when the stimulus of fever is no longer felt the heart fails from actual anæmia of its muscle. On the theory of the production of oligæmia we can understand why pneumonia should as stated by Huss be particularly dangerous to chlorotic females.

CHAPTER XVII

ACUTE FIBRINOUS PNEUMONIA—*Continued*

Treatment. Preliminary Remarks on Various Proposed Modes of Therapy.—There can be no greater confession of our inability to cope successfully with desperate cases of acute croupous pneumonia than is furnished by the long list of remedies proposed for its treatment. When we consider also that pneumonia is a self-limited disease we cannot fail to be impressed with the belief that the results claimed for certain modes of management may with propriety be attributed to the vagaries of the disease. It is to be feared that only too often the advocates of special remedies are misled by their enthusiasm and their desires.

During the many centuries in which pneumonia was conceived to be a local inflammatory process, bloodletting both local and general was so universally relied on that he was a bold man indeed who dared to oppose the method. When at length venesection was abandoned the pendulum swung to the opposite extreme, and he in turn became courageous who ventured to resort to this ancient practice. That the measure did not, however, deserve to sink into utter oblivion has been attested by the fact that men of recognized judgment and experience are again advocating the abstraction of blood under definite indications, as will be seen later on. Not so, however, with the administration of tartar emetic and veratrum viride which, as formerly employed, have deservedly fallen into disfavor.

The same may be said of the *quinine* treatment which, strongly indorsed by the elder Austin Flint and for a considerable time much in use, is now rarely if ever given because of its doubtful utility. Even while these pages are being written there have appeared in the journals some reports upon the quinine treatment of pneumonia which have attracted some attention. The use of this remedy is not at all new for it was extensively tried by German and English clinicians before the bacterial origin of pneumonia was clearly established. The rationale of its employment was based upon its antipyretic properties in large doses, 40 to 75 grains a day. The remedy was shown capable of lowering the temperature but, as proven by statistics, did not lessen the mortality beyond that of other modes of therapy. My opinion concerning the questionable efficacy of this drug has not been changed by the recent apparently favorable reports contributed to the medical journals.

Some six or eight years ago considerable interest was awakened by published reports of the favorable results obtained with two drugs, *creosote* and *salicylate of sodium*. The impetus in this direction appears to have been really imparted by a paper read before the New York Academy of Medicine in December, 1899 by Dr. Andrew H. Smith who is a practitioner of such wide experience and reputation that much weight was naturally attached to his statements. His view of the pathology of lobar pneumonia is that it is a local pneumococcus infection with secondary systemic disturbances caused by absorption of the toxins, that the germ thrives because it finds a suitable culture-medium in the exudate which it calls forth, and that the crisis occurs when the pabulum furnished by this culture-medium has become exhausted.

The diplococcus is a very vulnerable and short-lived organism and therefore he argues that if some remedy can be administered which, by being eliminated through the lungs can be brought in contact with and destroy the germ or antagonize its action, further baneful influence of the germ will be prevented and the disease will be limited to the time required by the pneumococcus for the exhaustion of the culture-medium already at its command. Such an agent Smith thinks is found in creosote and guaiacol or in their derivatives. On account of their easy administration and unirritating properties, therefore, preference is given to the carbonates of creosote and guaiacol. These are given in doses of ten or fifteen drops of the former and the same number of grains of the latter every two or three hours in order to bring the system under their influence as rapidly as possible. They are said to occasion very little disturbance and being broken up and slowly absorbed in the small intestines are held to maintain uniform antiseptic action upon the tissues to which they are carried by the blood-stream. No ill effects are said to be observed even when they produce their characteristic discoloration of the urine.

This treatment was not original with A. H. Smith but was first strongly advocated by him. It afterwards received the indorsement of several other physicians in different parts of the country, among them W. H. Thompson of New York City, W. O. Bridges of Omaha and Leonard Weber of New York, all of whom thought they saw very unpromising cases of pneumonia recover under the use of these agents in a way scarcely possible without them. Not only did the high fever fall within a day or two after instituting this treatment, but signs of resolution appeared and the course of the disease seemed to be shortened.

Such was the harmlessness of these two remedies that their trial appeared justifiable. Accordingly they were extensively used at first, but in the last two or three years have attracted but little attention. As before remarked, it is easy to attribute to medicine what may be merely the result of nature's efforts at resistance, yet it should always be remembered that pneumonia is a disease full of surprises.

If the reader decides to give these drugs a trial he may administer carbonate of creosote out of a spoon, in capsules or in the form of an agreeable emulsion, while guaiacol carbonate may be given in powders or capsules. The earlier they are begun in the course of the pneumonia and the more quickly the organism can be brought under their influence, the greater, it is claimed, will be the likelihood of success.

Salicylate of sodium is another remedy which, on the basis of seventy-two cases reported by Robert Liegel, is thought to exert a specific influence over this dread disease. Liegel's cases, if all were pneumonias, certainly make a highly gratifying showing. They occurred in persons between the ages of sixteen and seventy-four years. Eight are said to have had emphysema, six heart-disease and a considerable number to have been chronic alcoholics. Notwithstanding this unpromising material all are reported to have recovered. The salicylate was given in daily doses of 120 grains during the height of the malady and for several days after convalescence began. The great and, as it would seem to me, insurmountable objections to this agent are the gastric irritation and cardiac depression so apt to be occasioned.

Nitrate of silver is still another remedy, enthusiastically recommended by Caccianigra. Sixty cases with fifty-six recoveries, one slow convalescence and three deaths were reported by him in 1900. The silver salt is given to adults in daily doses of 0.25 to 0.30 (gm. 4 to 5) or one pill hourly of 0.02 to 0.05 until the maximum daily dosage has been reached. This treatment is continued until the temperature becomes normal and in some cases for a day or two longer. In very severe cases hypodermic injections are given of 15 c.c. of a 50 per cent solution of protargol.

Ergotin recommended by Kleckowsky is still another of the many remedies used against this formidable disease. From 50 to 75 grains are given daily in aqueous solution. If this treatment is begun early it is said to allay fever and to shorten the duration of the pneumonia, but if late, to quiet the respiration and diminish the degree of fever.

In the light of studies by Rosenow and others on the blood of pneumonia patients, it is strange that any hope can be entertained of the discovery of a medicinal agent that can exert a specific action. Furthermore, it is most difficult to judge of the real effects of any remedy in a disease which manifests such vagaries and surprises as does acute pneumonia. Consequently comment upon the modes of treatment mentioned above must be made with caution and reserve. Ergotin may possibly tend to counteract the effect of the pneumotoxins on vascular tension but the most I can say of these various drugs in the dosage recommended is that the patients require very tolerant stomachs and that *post hoc* is very likely to be confounded with *propter hoc*.

Antipneumococcic Serum.—The nature of acute pneumonia, its comparative brevity and its termination by crisis seem to place this malady in the category of those infections which offer a prospect of some specific mode of therapy. The well-known experiments of the Klemperer brothers with the blood serum of animals recovering from this infection aroused the hope that at last a remedy had been found. Accordingly pneumonia patients were given hypodermic injections of defibrinated serum obtained from animals and convalescent persons, but with very uncertain results. The efforts of experimenters were next directed to the production of an antipneumococcus serum.

Such a serum is now prepared by certain well-known manufacturing chemists and by at least one house is widely advertised as efficient against this dreaded malady. Hopes thus aroused are, however, not sustained by actual

results and by published figures. Thus G. E. Tyler is said to ha
141 cases of pneumonia with such a serum and to have had
deaths (Osler).

Tyler's figures are not as good as have been secured by other
therapy and I know of no series of authentic cases treated with se
furnish any better statistics. Consequently I must commend the
one prominent firm whose Chicago representative frankly state
reports his house had received from physicians in private and hos
tice did not warrant an unreserved recommendation of this remed

At present, therefore, it must be acknowledged that woefully
as are our weapons against this terrible foe to mankind we posses
mode of therapy than what is known as the expectant or sympto
of management. Before entering upon the discussion of this
treatment, it seems best to consider those measures which by some
are thought to exercise an appreciable influence in the way of al
the course of the disease.

Abortive Treatment.—The question is pertinent whether or not
ble to cut short this disease in its very inception. Opinion is
this point and I have always supported the negative side of thi
I believe that when pneumonia has appeared to be aborted the
was not in reality a pneumonia or was an instance of a one, two o
pneumonia as the case might be. The following instance illus
difficult a decision upon this point may be.

In the early Spring of 1900, a member of my own family who
quence of a multiplicity of duties, had become much run down w
to spend several hours superintending work in an unoccupied an
house. She was conscious of being not quite warm enough several
did not experience a decided chill until shortly before reachin
home. She then was seized with a profound shaking chill and f
difficulty in entering the house and ascending the stairs to her ap

I happened to be at home at the time and hearing some com
ceeded to ascertain its cause. I found the lady with chattering
shaking hands in the act of drinking a full dose of quinine and
for which she had called as soon as she entered the house. Beside
which had then lasted about twenty minutes she complained of a
in her right side just above the liver. I promptly took her tem
the mouth and found it registered 103° F. Her pulse was rathe
not noticeably accelerated, and respirations were about 28 or 30 a
considerable desire to cough.

She was at once gotten into bed, a large mustard plaster was
the entire base of the right lung and she received a heroin tablet o
of a grain and a teaspoonful of creosotol which had been hastily obt
a near-by pharmacy. I should state that I had previously exa
chest and recognized slight but distinct impairment of resonance i
infrascapular region and diminished respiratory sounds in this
This was about 6.30 P.M.

The patient passed a restless evening without any perceptible c toward midnight when her skin grew cooler, she fell into an unquiet sleep and her breathing grew deeper and much less rapid. She had expressed herself as feeling easier so soon as the sinapism began to burn which it did to such purpose as to blister. By two A.M. her condition was so manifestly improved that I ventured to seek repose. The next morning her temperature was a fifth or two above 100° F. and during the day declined to 99.8° F., she no longer experienced pain except from the effect of the mustard and did not cough.

Examination of the lungs showed no difference in the two bases as had existed the evening before. In the night, after I left her, she passed a large amount of light-colored urine. No further medicine was administered, she remained in bed, received light sustaining diet and on the second day her temperature at length became normal. She felt weak but on the third day was able to leave her bed.

I do not know what this attack was. It began with symptoms which were very suggestive of an incipient lobar pneumonia. I certainly believed it was pneumonia until the decline of fever after eight hours and general improvement threw me into a state of doubt. Some may declare this was an aborted pneumonia, but for my part I do not know. The lady, who had once before had pneumonia, declared that the onset in the two cases was identical.

The physician is not often fortunate enough to see a pneumonic patient during the initial chill, but if perchance he does, or if he arrives before the stage of engorgement has merged into that of red hepatization, he should at once resort to measures which, if they do not abort or restrict the extent of the pneumonic process, may at least mitigate the patient's distress. It is at this time especially that a hypodermic of $\frac{1}{8}$ to $\frac{1}{4}$ of morphine with $\frac{1}{150}$ of atropine is good practice. It soothes pain, quiets the patient and restricts his cough. The application of heat to the body and the administration of a hot draught may, together with the morphine, tend to flush the surface and shorten the duration or lessen the sense of chill. But this matter is difficult of determination inasmuch as the rigor is of brief duration, ordinarily, and is an expression of infection as shown by the fact of increased internal heat at the same time as the chill.

For amelioration of the pain a sinapism may be applied to the side or, as was done in von Ziemssen's wards at Munich, one may abstract a few ounces of blood by means of wet cups or by leeches. It was thought formerly that this lessened engorgement and, for the same reason, venesection was practiced. Under our modern conception of pneumonia as an infection, whether primarily local or general, it is difficult to see how any local and external treatment can exert an antiphlogistic effect or lessen the stage of congestion. If we would diminish the engorgement we must throttle infection in its inception and this we cannot do by venesection or any o means.

If venesection is ever justifiable in this *initial stage* it must be w there are signs of extensive and grave pulmonary œdema. Aufrecht rec nizes this as the only indication for venesection at this time and says he seen it do good under such circumstances.

An ice bag to the side or a hot poultice will often relieve the pain. A dose of calomel, grs. 3 to 10, should now be administered and may be followed in six to eight hours by a saline. This is not for the sake of any derivative action but to empty the bowels and stimulate a more active excretion and perhaps a better portal circulation.

If one is desirous of trying the effect of a so-called specific remedy, as carbonate of creosote or guaiacol, he should begin it now in this early stage that no time may be lost in getting it eliminated through the lungs. If it has any inhibitory influence over the pneumococcus, it should be brought into contact with the germ as soon as possible after this has begun its mischievous work within the pulmonary parenchyma. The remedy should be given in full doses and at short intervals, 15 to 30 drops of creosotol or an equal number of grains of the guaiacol carbonate every two hours. When the agent becomes apparent by its odor in breath or urine the dose may be somewhat reduced.

It is in this stage that veratrum viride was recommended on the ground that it diminished the engorgement by checking the blood-supply to the lungs. But I wish here and now to register a very emphatic protest against the use of veratrum in acute pneumonia. In the first place I believe the rationale of its supposed action is opposed to the modern conception of the pathology of this affection, the same as are other measures relied on to abort the malady. In the second place veratrum and allied cardiac depressants are dangerous and have unquestionably cost human life. The remedy is safe in the hands of a skillful physician who is alive to its dangers and keeps his patient under such close observation as promptly to stop the medicine as soon as signs of danger are detected or the engorgement has passed into hepatization. In the hands of an inexperienced practitioner or of the country doctor who is unable to visit his pneumonic patient more than once, possibly twice, in twenty-four hours veratrum viride and aconite are almost certain to do harm.

I shall never forget my horror when, early in my practice, I was told of a physician who prescribed veratrum for a man with pneumonia for the purpose, he said, of slowing his pulse. When, after a time, the pulse grew more rapid instead of more slow he ordered increased doses of the deadly drug. The pulse became still more rapid and feeble and the veratrum was again increased, and so on until, as was to be expected, the patient died, actually drugged to death by a doctor ignorant of the workings of the disease and of the drug.

It may be argued that because a remedy is capable of doing harm in the hands of the ignorant it is no reason why it should not be employed by the skilled. I maintain however that in the case of an agent whose beneficial action is open to doubt it is better to warn against its use altogether than to have it destroy a single human life, as in the wretched instance just cited. Therefore I make it an invariable rule to impress its dangers upon my students and to advise them strongly not to prescribe it at all in lobar pneumonia.

The disease under consideration is of itself sufficiently depressing to the heart without having that depression intensified by such a cardiac poison as veratrum, and when the exudate has once cut out from the circulation, as it were, a large area of the pulmonary system the right ventricle needs all the strength it can command. To continue the remedy is disastrous and to administer the drug in advance of this stage is to put the heart in a poor state to cope successfully with the foe when the time arrives in which it will surely need all its reserve power.

Prophylaxis.—The idea that the spread of acute lobar pneumonia in the community may be prevented, or that its contraction by individuals may be rendered less likely by the enforcement of certain sanitary regulations will probably strike both physicians and laity as chimerical. Yet the infectious nature of pneumonia and the identification of its specific cause render prophylactive measures not only rational but possible, within certain limits. Despite our boasted civilization and the marvelous advance in the last twenty years along the line of public hygiene, health boards have not yet been able to abolish the spitting habit nor even to arouse the people to its dangers. Indifference thereto is not confined to the laity but is widely prevalent, and far less excusable, among the mass of medical practitioners. Until physicians recognize and teach the dangers lurking in expectoration, no tangible results in the way of prevention of this and other pulmonary diseases can be expected.

By the foregoing remarks I do not wish to be taken as implying that the complete abolition of public spitting will eradicate pneumonia or tuberculosis from the community; but it should certainly contribute to the lessening of bacterial contamination of the atmosphere of public buildings and places of resort. For the present our efforts toward the prevention of croupous pneumonia must be limited to the hygiene of the patient and of his sick-room and to the toilet of the mouth and throat of the healthy.

Pneumonic sputa should be received in a vessel containing some antiseptic solution, as a 5-per-cent carbolic acid solution, and the sputum-cup should be carefully cleansed with boiling water several times daily. A bichloride solution is objectionable on account of its hardening action on the sputum.

If the expectoration is so sticky as to necessitate its removal from the mouth by a cloth, such pieces of cloth as are used for this purpose must then be burned. The patient's hands and mouth should be carefully washed whenever soiled by his expectoration and the nurse should cleanse the tongue and lips with a concentrated boric-acid solution or other efficient mouth-wash. Bedclothes and other articles soiled by the sputa should be sterilized with boiling water. The friends should be told of the infectious nature of the disease that they may not kiss the patient but may guard themse against undue exposure to infection by the sputum.

Wood's investigation into the viability of the pneumococcus in the mois coming from the mouth of the pneumonic sufferer, as previously mentio renders very pertinent the injunction against kissing and against the fri remaining in close proximity to the patient during coughing fits. Fur more, there must be no sweeping or dusting of the sick-room during illness, but the furniture, floor and walls should be wiped off with a damp cloth. Strict enforcement of such injunctions may render subsequent disinfection unnecessary, and yet such a measure is certainly wise, particularly if several cases of pneumonia have occurred in the same house.

Finally, although the rich bacterial flora of the mouth and throat cannot be destroyed by antiseptic sprays and gargles, still all persons in contact with a pneumonia patient will do well to attend strictly to the toilet of the mouth, throat and even nares. A simple cleansing antiseptic wash may be all that is required for this purpose. Moreover, during a season when pneumonia prevails all individuals should take especial care of their general health, and in particular should avoid dissipation, undue fatigue and exposure to the dangers of becoming suddenly chilled. Attacks of the *grippe*, colds on the chest, etc., must not be neglected and, in short, care must be taken not to lower vital resistance during the prevalence of this terrible scourge.

General Management.—As croupous pneumonia is a disease which tends to self-limitation the plan of treatment in highest favor is one which places the patient in the best possible state to resist until the affection has spent itself and convalescence sets in. To this end the vital forces must be upheld and dangerous symptoms combated. The fight is a defensive, not an offensive one.

In all cases, therefore, particularly when pyrexia is high and the infection is presumably correspondingly intense, it is of utmost importance to provide a competent nurse or nurses and to attend strictly to every detail of ventilation and nourishment that may anywise add to the chances of a successful issue.

Hygiene of the Sick-room.—In acute croupous pneumonia respiratory capacity is decreased, oxidation goes on rapidly and the blood is surcharged with the products of tissue-waste. Consequently the air supplied to the lungs should be as pure and as cool as possible. The windows should be kept widely open and the temperature of the room ought to be cool, the degree of coolness being determined by the height of fever. Even though the season be Winter and the atmosphere of the room become uncomfortably cold for the attendants, the windows ought to be open nevertheless. The nurses may wear warm wraps or a fire may be kept burning in the grate if such there be.

We have all seen pneumonia sufferers in rooms and wards so close and hot as to cause a person entering from the fresh air of the outer world to feel faint and half sick. How then can the patient burning with fever fail to have his condition rendered the worse by such an atmosphere! I often wonder if the high mortality of this disease in public hospitals be not, in a measure at least, owing to such unhygienic conditions. Can anything be more absurd than to see an oxygen apparatus in a room the windows and doors of which are closed and the atmosphere stifling from heat and foul emanations from patient and attendants. The notion that a pneumonia patient must be protected from cold air and draughts originated at the time when this affection was considered an inflammation of the lungs resulting from exposure. The modern view of its pathology renders ridiculous the fear that fresh, cold air is harmful. In my opinion, even a draught is less dangerous than is the lack of proper ventilation.

Light covering is another rule to be insisted upon in cases showing high fever; for, to heap blankets and comforters upon a pneumonia patient burning up with fever is equivalent to throwing fresh fuel on the flames one would suppress. The cotton jacket in particular is an abomination, a relic of ignorance and blind obedience to tradition, a device calculated to prevent radiation of body-heat, to augment the sufferer's discomfort, to retain sour-smelling sweat and to interfere with and hence to occasion neglect of those frequent chest-examinations without which one cannot keep track of the progress of the case.

As long, therefore, as the pyrexia is high there should be, at the most, a single light blanket and sheet, or only a sheet over the patient, while his chest is protected by simply a thin woolen shirt or, still better, in my opinion, a gauze undershirt, and over this a cotton or linen nightshirt. Such a covering interferes but slightly with radiation of body-heat and with chest examinations. All *clothing* worn by the patient should be changed daily not only for the sake of hygiene but also because it is a great comfort to the fever-sufferer to be put each morning into cool clean clothes.

The *diet* should be fluid and nourishing, there being nothing better than milk in some form or other. Seltzer or apollinaris water may be added to the milk when warm, after the manner of the Germans who, however, generally order Ems water. Thus alkalinized and charged with the gas of the water, milk becomes acceptable even to those who ordinarily are prejudiced against it.

Peptonized milk, kumyss, buttermilk, malted milk, albumen-water, which is water containing the white of egg, beef tea, made not from the ordinary beef extracts but from Mosquera's beef jelly, nutritious soups of various kinds, cocoa and chocolate are all serviceable and palatable foods which, in addition to possessing high food-value, supply to the system much-needed water.

In every acute infectious malady, especially one in which, like pneumonia, there is often great thirst, the patient *should be freely and often supplied with water.* In the febrile state, when tongue and throat are parched, the water should be cold though not iced. It is often very grateful to add some agreeable fruit juice as of lemon or orange to the water. The design of the fluids is not merely to quench thirst but to *flush the kidneys* and to eliminate toxins from the system. If nephritis supervenes the ingestion of large amounts of water becomes still more imperative.

The *mouth* should be cleansed daily with some agreeable antiseptic wash and the patient should receive a daily sponge bath, whether required by the temperature or not. If the bowels do not operate their action should be evoked by means of an enema and occasionally an aperient water.

Expectant Treatment.—The *fever* of sthenic pneumonia generally averages 103° F. or above and therefore calls for some measure for its reduction. Drugs that exert this effect are not to be recommended, chiefly because of their cardio-depressor action. Hare's statement that the coal-tar antipyretics diminish the leucocytosis, which is one of nature's safeguards, proves also a very practical objection to the use of these drugs.

Cold is now universally recognized as the best of all means for the control of pyrexia, and what holds good of typhoid fever and other infectious diseases applies equally well to acute pneumonia when characterized by high temperatures. Tepid sponging is said to have been employed by Hippocrates, but it was not until the middle of the nineteenth century that cold water became used by physicians in the reduction of the pyrexia of pneumonia. The followers of that ignorant yet practical empiric, Priessnitz, made cold applications to the chest, and in 1850 Traube advocated the use of cold affusions to the chest. Jürgensen was a strong advocate of cold bathing in pneumonia, particularly in children. The cold pack, even the ice pack, the ice cradle, cold compresses or the ice bag have all been tried and have their advocates.

The advantages of cold as an antipyretic are now so commonly recognized that it is a standing order in hospitals to subject all patients to this treatment whenever the temperature reaches 102.5° or 103° F. The method of applying cold now in most common use is sponging with tepid or cold water. Since the reduction of pyrexia by means of cold is practiced in other acute infectious maladies why should there be an exception in the case of acute pneumonia!

The main argument against antipyretic treatment in this disease lies in the consideration that the affection is usually short-lived and hence the pyrexia is less harmful than in some other infections. Such an objection is of less weight, however, than is the beneficial effect exerted on the nervous system and circulation. With reduction of the temperature by means of cold the patient becomes less restless and not infrequently falls into a refreshing sleep. This effect is especially noted in children. The pulse becomes stronger and slower and the respirations appear somewhat less rapid and painful. Delirium, when present in the fore part of the illness and due to pyrexia, is also favorably influenced.

Whereas the employment of cold in this disease cannot prevent complications and although death may occur in spite of energetic and systematic antipyresis, still, on the whole, statistics regarding mortality indicate a lessening of the death rate under this form of management. Statistics are often very deceptive, the excellent showing made in a certain class of cases being due to their favorable nature rather than to the special line of treatment. Yet it is scarcely reasonable to assume such a likelihood in the many hundreds of cases of pneumonia tabulated by writers which exhibit a far lower percentage of deaths than under any other mode of management.

Jürgensen sets this forth very clearly, as also did W. E. Ewart in his analysis of 1,000 cases of pneumonia treated at the London Hospital in the ten years ending with 1891. Whereas the mortality under a quinine treatment was about 40 per cent, and with the expectant plan of management was about 25 per cent, the mortality sank to 14 per cent in something more than 100 cases treated by means of cold.

A very effective means of applying cold and one that ought to be most grateful to a fever patient on a hot day is what Ewart designates as the "ice cradle," which can be constructed as follows: Two upright poles are placed on the floor and securely fastened one to the outside of the footboard at its center the other to the headboard. They are connected by a stout bar which thus passes in a line with the patient's body several feet above the surface of his bed (Fig. 29). A couple of sheets are then thrown over this ridge-pole and allowed to hang down below the side of the bed in such a manner as to form a tent and inclose the bed with its occupant (Fig. 30). From the horizontal pole are suspended five or six tin or, preferably, zinc pails filled with ice. These pails should be covered with thick felt or several layers of flannel cloth so as to catch the moisture caused by evaporation. The patient's body is then deprived of clothing entirely or in some instances is protected only by a thin cotton or linen gown and he is confined within this tent and subjected to the cooling influence of the atmosphere as it becomes chilled by the ice pails.

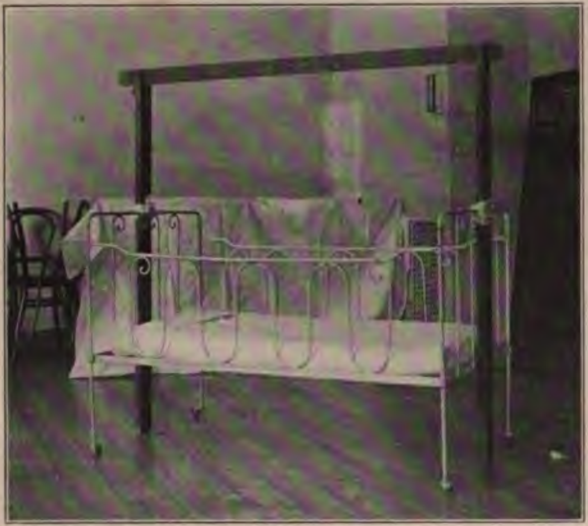


FIG. 29.—Framework for "ice cradle."

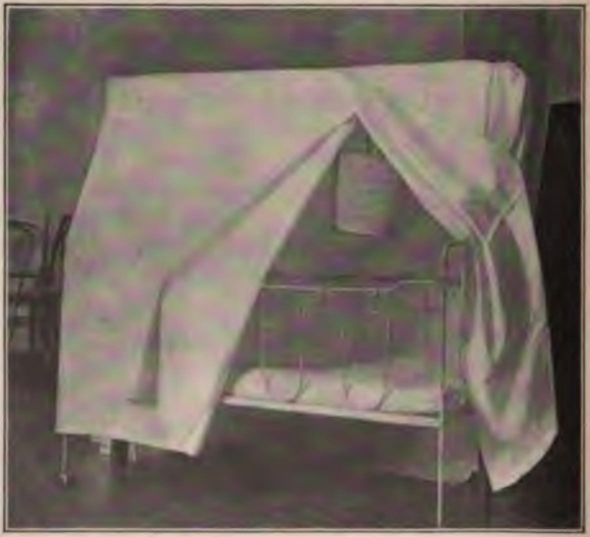


FIG. 30.—"Ice cradle" complete.

It is surprising how cold the air within the tent becomes even on a Summer day, there often being many degrees of difference between that inside and that outside the tent. In most instances the patient's fever begins to manifest a sensible reduction, but if this does not occur it may be inaugurated by an initial sponging. I once tried this on a lad of fifteen whose temperature of 105° F. before was easily maintained below 102° F. after he was placed inside this cheaply improvised cooling-room.

If at any time the body-temperature falls to a point that occasions a feeling of chilliness, the pails of ice may be removed for a time or, what is better, a light covering may be thrown over the patient. This means of applying cold is more effectual and continuous than sponging and on a hot Summer day affords a grateful relief from the heat of the atmosphere. There is no danger to be apprehended from the patient's taking cold which appears to be a veritable black beast to the public; but should his feet become cold they can be warmed by a hot-water bottle without counteracting the antipyretic action of this cold-air bath.

The favorable effect of the ice cradle is an additional argument for having the pneumonic patient thinly covered, as was insisted upon earlier in this section on treatment. It also emphasizes what was said regarding the keeping of the sick-room cool. Indeed, the same effect may be accomplished if, as was done in the case of some of Ewart's hospital patients, the pneumonia sufferer burning with fever be almost uncovered and then exposed to the cold invigorating air of a room in which the windows are kept open. He is thus enabled to breathe pure cool air while at the same time his temperature is being reduced by radiation.

The cold bath is another means of lowering temperature which is warranted in cases showing hyperpyrexia, and which, in children at least, has the emphatic indorsement of no less an authority than Juergensen. It is difficult of execution in adults and would probably aggravate their pain and dyspnœa in consequence of the change of position entailed. This would be a serious drawback to the bath when the heart shows signs of feebleness and hence an ice-rub is preferable and equally efficient. Young children may be lifted into a tub without subjecting them to much discomfort, and in cases indicating antipyresis the cold bath will be found to yield excellent results.

The practitioner who finds an antipyretic treatment indicated need not be deterred by the prejudices of friends or neighbors. He will generally find that a simple frank explanation of the objects to be gained will remove, or at least silence, their objections. At all events such has been my experience.

The foregoing advocacy of a judicious yet energetic use of cold in acute pneumonia is not to be taken as constituting the means on which reliance is chiefly to be based. It is only a means to an end and this end is the lessening or removal of injurious symptoms and the support of the vital powers until nature comes to the patient's rescue in the form of crisis. We will now pass therefore to the consideration of other symptoms calling for special attention.

Pain is not a symptom which, in general, requires special treatment. In the beginning, as already advised, a hypodermic of $\frac{1}{8}$ or $\frac{1}{6}$ occasionally $\frac{1}{4}$ of morphine may be given with advantage, excepting, of course, to the aged and to very tender children who do not bear opiates well. It is not usually necessary or advisable to repeat the morphine because of pain, lest it blunt too greatly the patient's sensibility. Should pain call for special attention the movements of the side may be restricted by strapping with strips of adhesive plaster as will be described in considering pleurisy. Decided relief is sometimes found to follow the application over the affected lobe of either an ice bag or a flaxseed poultice, with or without mustard. My preference is for the ice bag, which can generally be tolerated if there be a thin layer of muslin between the bag and the skin. Furthermore, if fever is high the poultice seems to me objectionable on grounds that have already been stated with reference to the cotton jacket.

Cough is a symptom unavoidably present and of varying degrees of frequency and severity. It occasions more or less pain and is therefore restrained by the sufferer so far as possible. It is best not to prescribe sedatives for this symptom unless it robs the patient of needed rest. For this purpose heroin in $\frac{1}{12}$-grain doses at six to eight hour intervals or codein in $\frac{1}{2}$-grain doses are preferable to morphine. Expectorants are of no service for this symptom and are worse than useless.

Insomnia is another symptom that may or may not call for treatment. If the patient show persistent wakefulness there is danger of exhaustion and a safe hypnotic must be administered. Paraldehyde, sulphonal, trional, chloral together with a bromide are all reasonably safe so long as cardiac asthenia is not threatening. In such a case morphine hypodermically is best. Marked insomnia is a result of the infection or the effect of the pyrexia acting on the cerebral centers. It is, therefore, most intractable in some cases. In such instances a short but refreshing nap may follow the use of the cold bath in children or an ice-rub in adults.

Headache is sometimes so severe as to call for special measures for its relief. An ice-cap is generally very grateful and should be tried. In other instances a mustard plaster at the nape of the neck mitigates the suffering. When such simple measures fail then it may be necessary to try bromides or some of the less depressing but distinctly analgesic products of synthetic chemistry, as kryofin and phenacetin. Such agents ought, however, to be administered with caution and only employed after the powerlessness of other simple means has been proven.

Delirium.—This symptom has different degrees of significance according to the time of its occurrence and its violence. Coming on early and when the fever is high it is a result of the pyrexia and may be overcome by the measures advised for the reduction of the temperature. When, on the contrary, delirium appears after a number of days and shortly before the hoped-for crisis it is due to exhaustion, is often extremely violent and of evil omen.

This *exhaustion delirium* must be quieted if possible by the exhibition of stimulants. Fraenkel recommends a hypodermic injection of morphine, $\frac{1}{8}$ to $\frac{1}{4}$ grain depending on the case. Care is called for in the use of morphine if there be extensive bronchitis or pulmonary œdema. Hydrobromate of hyoscine $\frac{1}{100}$ hourly, by the mouth or under the skin, is also useful. If fever be also high I would recommend an ice rub. Patients with delirium must be closely and unremittingly watched, even strapped to the bed, to prevent them from jumping from the window or otherwise doing themselves harm.

Postcritical delirium, which is likewise an exhaustion delirium, seldom requires special treatment. It is likely to disappear as convalescence progresses. Nourishment and stimulants are the best treatment for this kind of delirium, although morphine judiciously administered is also good.

Diarrhœa, particularly septic diarrhœa with its horribly fœtid and frequent stools, is a complication that often baffles medical skill. As soon as the abdomen begins to show tympanites and the discharges commence to be offensive a full dose of calomel should be given so as to clean out the upper bowel. High saline enemas should then be administered every two or three hours and the patient should receive by mouth large doses of salol, subgallate of bismuth, benzonaphthol, or other drugs supposed to exert an antiseptic action in the small intestine. At the same time the food should be so modified as to exclude articles which, being of animal origin, are prone to putrefactive decomposition. The diet at this time should consist preferably of cereals, rice-water or barley-water, etc. The patient should also be made to drink large quantities of hot sterilized water.

Tympanites is another condition which, although often present in a minor degree, sometimes becomes extreme and embarrasses respiration. It is probably an indication of the paralyzing action of the toxins on the coats of the bowel. For this reason attempts at its removal are apt to be futile. Nevertheless, as I have seen patients recover in whom distention of the abdomen was a pronounced and distressing feature, I would urge resort to energetic measures for its subsidence. Hot fomentations, stimulating applications of mustard or turpentine, asafœtida in large doses either in pill form or as enemata of the infusion, massage, or the faradic current may be tried.

Heart-weakness.—The danger that threatens the pneumonia sufferer is on the side of the circulatory system. Doubtless, as has been dwelt on before in these pages, there are several factors at work in bringing about heart-weakness, among them being strain on the right heart resulting from mechanical interference with the blood-flow through the hepatized lung. That such is not the only nor even the chief factor is shown by the occurrence of fatal heart-weakness in cases presenting very limited consolidation and, on the other hand, by recovery in cases characterized by hepatization of several lobes.

The only conclusion to be drawn from such considerations is that the asystolism is the expression of the infection acting on the myocardium either directly or indirectly. The experiments of Romberg and Paessler, to which reference has been made in previous pages, indicate that in some instances the principal effect of the poison is expended on the vaso-motor centers and that the failure of the heart is due largely to the capillary paresis resulting from the action of the toxins on the vaso-motor centers.

In cases of this kind cyanosis becomes extreme and makes its appearance prior to appreciable evidence of cardiac incompetence. There are other cases, however, in which evidence of cardiac debility appears before there is marked cyanosis, and in these it is a fair inference that the toxins exert their main action on the myocardium.

In these cases the heart-weakness is shown by an *increasing frequency of the pulse* and steadily lessening intensity of the heart-sounds, in particular of the *pulmonic second* tone. Coarse bubbling râles are apt to appear and then evince growing stasis within the pulmonary circuit, although such a condition is not necessarily present until at the very termination of the struggle. In a few instances pulmonary œdema sets in abruptly and is the immediate cause of death. The precise origin of this œdema is not yet established but in some cases it is probably in cardiac inadequacy.

In the light of the foregoing facts, what are we to do to ward off or to overcome the effect of the toxæmia on the circulatory apparatus? Are we to wait until dangerous signs appear and then endeavor to counteract them, or shall we institute a treatment early that may forestall and prevent circulatory failure? To my mind a waiting policy is like locking the barn doors after the horses have been stolen. Over and over again I have been hurriedly called in consultation to see a pneumonia patient after the rapidity of the heart's action or other signs had excited the physician's or the family's alarm. Under such circumstances medical skill is usually powerless; the mischief has been wrought already and whatever may be done should have been commenced hours, nay days earlier.

The only weapons we possess against this enemy of the pneumonia patient are *digitalis* and other *heart tonics* and *stimulants.* These will now be considered at length and in detail. Of late my experience in acute pneumonia has been strengthening my conviction that in digitalis we have a potent means of defense against the weakening effect of the poison on the myocardium and possibly also against their paralyzing influence on the vaso-motor centers. Doubtless there are cases of such malignancy that the patients may be said to be doomed from the outset, and in such nothing is of avail; but in most instances time and opportunity are afforded for a vigorous fight.

The **digitalis treatment** of pneumonia is not by any means new but dates from the writings of Rasori and Traube. The latter employed the drug chiefly as an antipyretic, but in so doing observed its effect on the heart and called attention to the fact, since abundantly confirmed, that pneumonia patients tolerate far greater doses than do cardiopaths. As a febrifuge Traube prescribed a total dosage of 30 to 60 grains, while in pneumonia he recommended as much as 100 to 120 grains administered in the course of several days.

Within recent years it is Petresco of Bucharest who has called the attention of the profession to the digitalis treatment of acute pneumonia, advising doses which astound and terrify most physicians. Such are the remarkable results claimed by Petresco and such the tolerance displayed by his patients that writers commenting thereon have been inclined to attribute these to the age and vigor of his patients (these being soldiers) and to some peculiar quality of the plant from which his digitalis was obtained.

Regarding the former explanation it may be urged that although pneumonia is less fatal among young robust persons as soldiers, still 1 to 2 per cent of deaths is a strikingly low death rate, even in a military hospital. Concerning the latter assumption that some peculiarity exists in Petresco's digitalis it may be replied, Petresco's experience is not exceptional but accords with the statements made by Traube many years earlier. Moreover, his claims have been supported by a number of other observers, as Lépine, Huchard and Rendu in France, Masius in Belgium and Fraenkel in Germany. Lop, presumably a Frenchman since his contribution appeared in the *Revue de Medicine*, 1895, and Balfour have also advised digitalis in heroic doses in this disease, the former in daily doses of 10 grammes (150 grains) and the latter in ½-drachm doses of the tincture in conjunction with hydrate of chloral.

Petresco exhibits 4 grammes or more (60 or more grains) daily for a period of four to five days, until the patient has received in all 20 to 24 grammes (300 to 360 grains). These enormous doses are administered in a watery infusion and are said to cut short the pyrexia and even to cause a remarkably brief duration of the disease. Unfortunate effects are never observed. Such heroic, nay huge doses might be thought dangerous were it not for the testimony of others who declare digitalis to exert a very different effect in cases of acute pneumonia than in heart disease and to be far less dangerous.

Thus Fraenkel states that for several years prior to the publication of his work in 1904 he has been employing digitalis in a similarly energetic manner and is convinced of its ability to prevent the alarming and abnormal acceleration of the pulse so often seen in acute pneumonia, and that also it exerts a marked influence in maintaining blood-pressure.

Fraenkel's method of its administration is as follows: 3 to 4 grammes of the crude drug (45 to 60 grains) are administered daily for three days, a total dosage of 12 grammes (180 grains) being never exceeded. Presumably this amount is exhibited in a watery infusion by mouth, since Fraenkel states that when nausea and vomiting occur he gives the remedy by rectum, injecting four times daily 25 c.c. (approximately 3 drachms) of a 4-per-cent aqueous infusion, which would equal a total daily amount of 100 c.c. (approximately 3 ounces) containing 4 grammes (60 grains) of the digitalis leaves.

Traube pointed out the fact that digitalis exerts a far more powerful effect on the pulse toward the close than at the beginning of pneumonia and therefore Fraenkel never prescribes the drug after the third day of the illness. Furthermore, he considers it contra-indicated in persons with heart disease, chronic nephritis or arteriosclerosis or those who are drunkards or past fifty years of age.

Administered in this manner digitalis begins to lower the temperature after twenty-four hours, the decline being steady or by drops. The pulse, moreover, instead of becoming more frequent as would be the case were the effect of the remedy toxic, shows a tendency to fall from ten to twenty beats per minute. A more decided reduction in the pulse-rate does not take place until two or three days after discontinuance of the remedy, and after the temperature has returned to normal. Never has a fall in blood-pressure been observed but instead Gaertner's Tonometer has demonstrated a tendency to rise of pulse-tension, particularly in cases in which at first this was low. In Fraenkel's opinion, digitalis possesses no specific influence over the inflammatory process but merely causes defervescence sooner than would otherwise be the case.

It must be confessed that such heroic doses of digitalis impress one at first thought as simply poisonous and as subjecting the pneumonia patient to an added and even greater danger. Yet, I must admit that there are several considerations in favor of this method of treating suitable cases: (1) The opinion of men of experience and judgment is entitled to due weight. (2) The rationale of the treatment seems to fulfill the indications, namely, to sustain the heart and maintain blood-pressure while at the same time reducing temperature. (3) In so stormy and often so fatal a disease against which we possess no specific remedy any plan of management is justifiable which seems devoid of harm provided proper caution is exercised.

For the assistance of any physician of the United States who may desire to make trial of this digitalis treatment as carried out by Petresco and Fraenkel the following information is furnished. An infusion of the leaves may be prepared of the strength used by Fraenkel, or it may be of the strength prescribed by the United States Pharmacopœia which is 15 grammes of English leaves to 1,000 c.c. of water. Of this latter infusion 210 c.c. or 7 ounces equal 45 grains, while 240 c.c. or 8 ounces are equivalent to 60 grains of the crude drug. This amount may be administered daily for three or four days, *provided the patient come under treatment inside the first three days dating from the initial chill, and provided furthermore that no contra-indications exist in heart, blood-vessels, kidneys, age or habits.* Taken ice cold the infusion is not likely to disturb the stomach, but if so may be given by rectum. Should a fresh and reliable infusion not be obtainable, then the tincture or fluid extract may be prescribed in doses equivalent to the amount of crude drug required, or a preparation presently to be mentioned may be administered hypodermically.

The strength of a reliable fluid extract is such that 1 minim equals 1 grain of the leaves and accordingly 45 to 60 minims of this preparation would be the daily dosage. My preference over the fluid extract is for the fat free tincture because it is of reliable strength and does not disturb the stomach. Of this tincture 10 drops (7 minims) are equivalent to 1 grain of the leaves, and therefore from 450 to 600 drops (315 to 420 minims = 5 to 7 drachms) would have to be administered daily for three days.

This and other tinctures of official strength, U. S. P., are stronger than those that may be prepared henceforth in accordance with the eighth revision of the U. S. P., issued in September, 1905. Therefore, if the tincture employed be of the strength advised by the eighth revision, larger doses must be given since 15 drops correspond to 10 drops as tinctures were formerly made.

There is a serious objection to the exhibition by the mouth of such large doses of any digitalis preparation, namely, the disagreeableness of the taste which makes it very trying even when it does not disturb the stomach. On this account it would be very desirable if the remedy could be administered under the skin. There is a preparation known as digitalone which is made especially for hypodermic administration and which I have employed many times. Some individuals complain of pain at the site of injection and a little redness and swelling are apt to remain for a number of hours. On this account it is likely that considerable discomfort would attend the injection of such large amounts as would be necessary. Nevertheless digitalone may be tried and will not be likely to occasion intolerable suffering if the injection be made slowly and be succeeded by massage of the part and by the application of a wet boric-acid dressing.

The strength of digitalone is the same as that of a reliable tincture prepared in accordance with the directions contained in the seventh revision of the United States Pharmacopœia, and accordingly 10 drops or 7 minims represent 1 grain of the leaves. Therefore, if digitalone is to be employed it will have to be injected in doses of 5 to 7 drachms daily in order to obtain the effect desired. This amount would best be injected in relatively small quantities often, and hence 30 minims or a syringeful every two hours would be necessary.

Such doses are positively startling but are based on statements made me by two reliable pharmaceutical chemists whose preparations I am in the habit of using. I have had no experience with digitalis in these large amounts, but shall not hesitate to give this plan of management a trial in the first case coming under observation which furnishes the indications laid down in the preceding paragraphs. Justification for such treatment is found in the statements of such clinicians as Traube, Fraenkel, Petresco and others that digitalis is tolerated by pneumonia patients in vastly larger doses than would be safe in persons with heart disease who, as a rule, constitute the class of invalids for whom this invaluable remedy is prescribed.

Other heart tonics and measures for use when digitalis is contra-indicated. The query naturally arises, what are we to do when a pneumonia patient is not seen until after three days, or after the time in which it is admissible to institute the digitalis treatment, and what is the management suitable when the attack occurs in the senile, the alcoholic or the individual with chronic renal, arterial or cardiac disease? These are the cases so commonly seen in the hospital, which swell the mortality records of this terrible malady. The pulse may be unduly rapid from the outset or it may all at once begin to increase in frequency, going up steadily hour by hour, from 120 to 130, 140, 150 or more and at the same time may become thready or possibly intermittent. The heart-sounds too are of increasing feebleness, while the pulmonic second tone in particular loses strength and clearness.

Caffeine, in my opinion, is the drug *par excellence* on which reliance must be placed. The remedy is best administered hypodermically and in full doses. From 1 to 3 grains may be given depending on the urgency of the case. In the beginning it may be administered every four hours and in doses of 1 or 2 grains, the strength and frequency being increased as the disease advances if, in the judgment of the practitioner, this becomes advisable. Caffeine and its salts are not very soluble and there is much want of knowledge concerning the various preparations on the market both as to their solubility and their percentage of the alkaloid. Accordingly, I have made inquiries of a reliable chemist and have ascertained the following facts.

Caffeine citrate, which is a citrated caffeine and not a true salt, is soluble in 4 parts of water but is precipitated by the addition of another part of water. When however 25 parts of water are added to citrated caffeine a clear and stable solution is obtained. Accordingly, 1 grain should be dissolved in 25 minims of water. *Caffeine sodio-benzoate* is the next salt most commonly found in the shops but is weak in the percentage of alkaloid, being a mixture of 50 parts of caffeine with 59 of sodium benzoate. It is soluble in the proportion of 1 to 7. It is convenient, therefore, by reason of its solubility, but one should remember that in prescribing 1 grain he is really giving only half a grain. *Benzoate of caffeine*, soluble in 20 parts of water, contains approximately 63.5 per cent of the alkaloid and is a true salt. *Valerianate of caffeine*, soluble in 30 parts of water, is also a true salt and contains approximately 67.5 per cent of the alkaloid.

From the foregoing facts it is seen that if caffeine or one of its salts is to be administered in doses of 1 to 3 grains it will necessitate the injection of considerable amounts of fluid. Therefore I would advise the formula recommended by the chemist I consulted; namely, caffeine 5 grains, sodium benzoate 8 grains, water 50 minims of which 10 minims contain 1 grain of the alkaloid. Even of this it would be necessary to inject 30 minims in order to give 3 grains, and such a quantity is apt to occasion slight pain.

Nevertheless, such is the value of caffeine in sustaining the power of the heart without increasing its rate that it is indispensable in these cases. Hot coffee has long been employed as a heart stimulant in pneumonia but is inferior to the alkaloid. It may be given by mouth or as an enema.

Finally, I may state that in a desperate case I should not hesitate to employ small hypodermic injections of digitalone in conjunction with caffeine, e. g., 10 to 20 minims once in six hours, if pulse-tension be very low and the heart be showing alarming increase in its rapidity.

Strychnine subcutaneously is another agent more in use than any other single drug for the treatment of pneumonia. It is far inferior to caffeine or digitalis, and on that account is not nearly so often employed in England and on the Continent as in the United States. For my part, I am becoming very skeptical of the advantages of strychnine as a heart-tonic and I am quite sure that in its use to the exclusion of other remedies, or in the large doses often prescribed, this medicine does harm rather than good. True, it is said to sustain pulse-tension, but the effect in this respect is inferior to that of caffeine and of two other agents now to be mentioned.

Camphor is another cardiac tonic of known power and efficiency which is in far greater use abroad than here. The drug exerts a promptly stimulating effect and on this account is of great service when the heart shows signs of flagging, the pulse becoming thready and even intermittent. The action of camphor is not sustained, however, as is that of digitalis, while, unlike caffeine, it increases the frequency as well as the force of the pulse. The agent is best administered by injecting beneath the skin 15 to 30 minims of a 10-per-cent solution of the gum in sterilized olive oil.

Such a dose may be given in conjunction with caffeine, that is, every four or two hours, as the case may require, so as to alternate with the injections of the caffeine salt; or as often as every hour if the case be very threatening. The benefits to be derived from such frequent dosage of camphor over a period of many hours or even of one or two days will offset any deleterious effects on the nervous system or renal excretion.

Musk is a third stimulant of the heart on whose power and reliability I desire to lay particular stress, and which deserves to be used in the heart failure of pneumonia very much more than is the case in this country. One reason for its neglect in the United States is its cost, on which account perhaps it is apt to be adulterated. The retail chemist not infrequently charges as much as $1.50 or $2.00 a grain, a price that is wholly unwarranted for, as I am assured by a manufacturing pharmacist of Chicago, pure musk can be obtained in original packages for $4.00 a drachm.

S. G. Bonney, of Denver, has informed me of his having administered as much as 15 grains by mouth at a single dose and repeated this amount in four hours. He says he believes he has seen it save life in cases of pneumonia when all other stimulants had proven powerless. Such method of administration is inferior, however, to the hypodermic injection.

The only official preparation of musk that can be exhibited subcutaneously is the tincture, and of this 15 minims may be injected as often as occasion demands. Musk is soluble, however, in distilled water, without the addition of alcohol (1 grain to 5 minims), or a solution may be prepared as follows:

℞ Musk	1.0 gm.
Alcohol	1.0 "
Borax	0.5 "
Water	ad 15.0 gms.

of which the dose is 15 minims. I know from personal experience that musk hypodermically is a positive heart-tonic which acts promptly and maintains its effect for at least two hours.

The three drugs of which mention has been made, namely, caffeine, camphor and musk, will often do wonders in rallying and sustaining a failing heart, as was manifested by the following case which I saw daily in consultation with Dr. J. A. Capps of Chicago in the Spring of 1905.

Mr. E. K., a banker of sixty-five, had been confined to the house and most of the time to his bed for more than a month. The illness had been a right-side pleurisy of pneumococcus origin that had set in nearly six weeks before my visits began. Of previous illnesses it is only necessary to say he had a well-marked mitral stenosis which dated from boyhood, and because of which, probably, he had always been a delicate man. Habits were exemplary.

The patient was first seen by me Monday at about noon. He was in bed and displayed signs of pleuritic exudate in the lower portion of the right thorax which did not appear to call for aspiration. His temperature was not much above normal, his pulse and respirations were satisfactory. In fact, such was his gain in strength during the week just ending that it was advised to take him to a mild climate where he could remain out of doors.

The following morning, Tuesday, his condition was worse and it was possible to make out a small patch of dullness higher up, with associated signs and symptoms that made it clear that a pneumonia was present.

By the late afternoon the increasing pulse-rate indicated a heart-tonic and 10 minims of digitalone were ordered hypodermically every four hours. It is superfluous to say the hygiene of the sick-room and the nursing were of the best. At the time of ordering the digitalis preparation the pulse was about 110, having gone up a number of beats since morning. Its quality was not good and this, together with the mitral lesion, occasioned great uneasiness.

During the night, and after only three doses of the digitalone had been given, blood-pressure fell from 108, with the Riva-Rocci broad arm band instrument, continuing to decrease in spite of the injections until, at the hour of my visit, Wednesday, 9 A.M., blood-pressure could not be recorded and the radial pulse was absolutely imperceptible.

Auscultation of the heart showed that it was beating not far from 100 times a minute, that the presystolic murmur was barely audible, that the first sound at the apex was very short and sharp, and that the second tone at the apex could be scarcely distinguished, while the pulmonic second was ringing and still loud. Percussion showed enormous distention of the left auricle, deep dullness reaching fully an inch outside the vertical nipple line and equidistant with the outer margin of the ventricle at the level of the nipple.

The patient was conscious but lethargic and in a cold perspiration, despite a temperature of about 102° F. in the rectum. Here then was a very desperate condition which, in my opinion, portended a fatal termination before very many hours.

What was the explanation? Had the digitalis overpowered the heart or was the condition the effect of the toxæmia? Leucocytes were 55,000 and hence general resistance seemed good. The fact that the pulse-tension had fallen early, to 60 soon after midnight and after only 30 minims of the drug had been administered, seemed to indicate that the overdistention of the left auricle and emptiness of the ventricle were not the result of the digitalis.

Whatever was the explanation, it was plain that the man's only chance of recovery lay in some means of unloading the auricle and the right heart, and of reëstablishing a discharge of blood into the aortic system. Doubtless the immediate abstraction of blood from the arm would have lessened venous stasis and would have helped to reinstate the arterial circulation. On a similar occasion this is what I should advise, but, as it was, the following orders were given:

Stop digitalis at once and resort to frequent stimulation with caffeine, camphor and musk, in accordance with the directions given in these pages. The musk was to be given by mouth, and in addition ammonia was to be administered, 15 drops of the aromatic spirits in water.

By twelve o'clock the patient's condition was practically unchanged, owing, perhaps, to unavoidable delay in beginning vigorous stimulation and to his having been unable to retain the musk in capsule form. Accordingly it was decided to resort wholly to hypodermic medication and in the following manner: One grain of caffeine benzoate, 15 minims of camphorated oil and 1 grain of musk were each to be injected every six hours in rotation so as to insure the administration of one of the stimulants every two hours. Ammonia or a little brandy was to be added internally, if occasion demanded, and nourishment in the form of hot milk or broth was not to be neglected. Another and, to my mind, indispensable provision was the constant attendance day and night of the medical practitioner as long as danger existed.

These directions were carried out faithfully and at my visit at 5 P.M. I was overjoyed at being able to detect a slight flicker in the radial artery. The patient's mental state had become natural and his skin had lost the clamminess observed in the forenoon. At nine o'clock in the evening all of the pulse-waves could be counted at the wrist and blood-pressure had risen nearly to normal.

Moreover, a striking change had taken place in the cardiac findings. On percussion it was apparent that the outer border of the left auricle had receded toward the median line, while palpation detected a distinct moderately long presystolic thrill, and on auscultation the corresponding murmur, nearly obliterated in the early morning, could be perceived distinctly. It was plainly evident in all ways and to all attendants that an almost miraculous improvement had taken place and the patient himself expressed his consciousness of the same.

On the following morning pulse-tension was 108 mm. and everything was progressing to our entire satisfaction. During the progress of this case Dr. Capps made a series of highly interesting and instructive observations to determine, if possible, the effect on blood-pressure of musk, caffeine and strychnine. At 2.10 P.M. blood-pressure was 112 mm. and at 2.11 P.M. 5 minims of the musk solution containing 1 grain of the drug were injected. At 2.17 P.M. blood-pressure had risen to 119, at 2.21 P.M. it was 115, at 2.27 P.M. was 115, at 2.36 P.M. was 114, at 2.56 P.M. was 113, at 3.10 P.M. was 112 and at 3.45 P.M. was still 112, the same as at the beginning (Fig. 31). At 3.46 P.M. strych. sulph. $\frac{1}{30}$ was injected and at 3.50 P.M. blood-pressure was still 112; at 3.55 P.M. it was 113, at 4 P.M. it was 115, at 4.20 P.M. it was 113,

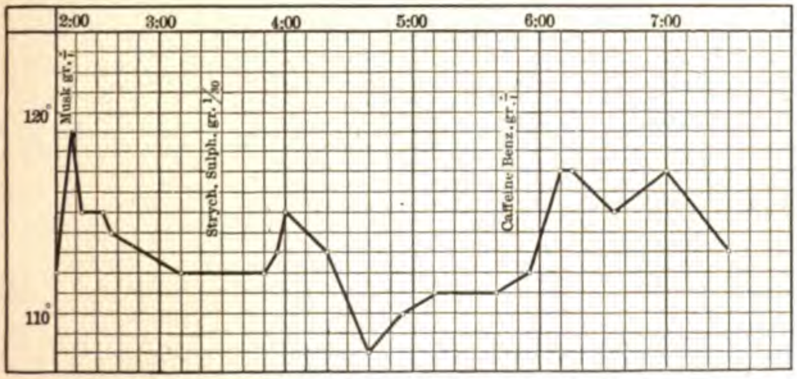


FIG. 31.—Chart showing variations of blood pressure induced by stimulants.

at 4.40 P.M. it was 108, at 4.55 P.M. it was 110, at 5.11 P.M. it was 111 and at 5.40 P.M. was 111. At 5.41 P.M. a grain of caffeine benzoate was injected and at 5.56 P.M. blood-pressure was 112, at 6 P.M. it was 117, at 6.15 P.M. it was 117, at 6.35 P.M. it was 115, at 7 P.M. it was 117 and at 7.30 P.M. it was 113. The foregoing observations are not at all conclusive. Moreover, there is a possibility of error in the determination of such slight differences. For instance it is difficult to explain the drop of 5 points in the instance of the strychnine from 113 at 4.20 P.M. to 108 at 4.40 P.M., and then its rise to 110 at 4.55 P.M. and 111 at 5.10 P.M. Also, in the case of the caffeine, the fall from 117 at 6.15 P.M. to 115 at 6.35 P.M. and its subsequent rise to 117 at 7 P.M. was puzzling. There may have been some additional factor, as a hot drink, that was not noticed by the doctor. Nevertheless, the observations show that the effect of the musk was more prompt, more pronounced and more sustained than that of the strychnine. The effect of the caffeine also was more pronounced and sustained than that of the strychnine and was sustained for a longer time than that of the musk.

I regret that similar observations were not made with respect to the influence of camphor, but am certain that it would have been found very positive on blood-pressure since the finger shows so plainly that it increases the force of the heart-beats.

While dwelling on this subject of the effect of therapeutic agents on blood-pressure it may be stated that Dr. Capps has also kindly furnished me with a record of the action of whisky and ammonia administered by mouth. The case was not one of pneumonia but of pernicious anæmia with attacks of tachycardia that were relieved by the ingestion of 2 ounces of whisky and 15 minims of ammonia. But their power in lowering pulse-tension is very pronounced.

At 11.30 A.M. blood-pressure was 164 and pulse-rate was 175. At the same time the two agents mentioned were swallowed and at 11.50 A.M. blood-pressure was 82 and pulse-rate was 150; at 12.10 P.M. pressure was 80 and rate was 150, and at 1 P.M. pressure was 100 but the rate had fallen to 80.

The very striking reduction in blood-pressure in this case leaves no reasonable doubt of the accuracy of the reading and proves what will be said on the inadmissibility of alcohol in many cases of pneumonia.

In the case just narrated the heart-tonics were administered, one or another, at two-hour intervals. But such need not by any means be the limit of stimulation in desperate cases. For the purpose of tiding a patient over some hours or even a few days of peril one need not hesitate to inject these and other remedies as often as every hour and in very much larger doses than were used in this case. It goes without saying that the *indications for heart stimulation* are to be found in careful study of the pulse, *not alone in its frequency but in its force and volume.*

The initial indication for the institution of vigorous stimulating treatment may reside in the rate and progressive increase of the frequency of the pulse, but the energy, frequency and continuance of the stimulation must be determined by the force, volume and rhythm of the pulse. The skilled physician, who, by years of careful practice, has acquired the *tactus eruditis,* may b able to appreciate the quality of the pulse by the unaided finger. For the most part, however, such nicety of observation is a lost art, and hence the physician would do well to employ a sphygmomanometer with which to register pulse-tension, since in this lies the truest means of determining the effect of cardiac stimulants and of their persistence.

If, despite the use of camphor, caffeine and musk, the pulse-rate continues to rise, or any augmentation of its strength passes away quickly, they are not doing permanent good and they must be increased in dose or frequency of administration. Should these agents perchance be already pushed to the limit of their usefulness, then it may be well to fall back on other stimulants, though not to the exclusion of the ones already in use.

Ammonia is a powerful but very evanescent stimulant to the heart. It should be given, therefore, not in large doses at considerable intervals, but in *smaller amounts and often.* Five grains of the carbonate in mucilage of gum arabic without sugar every half hour or twenty minutes, the aromatic spirits in 15- to 30-drop doses at the same interval, and the German liquor ammoniæ anasatus in the same dosage are all efficient, but they are all open to the objection of irritating the stomach when long continued. In an emergency, as sudden collapse, the aromatic spirits may be thrown under the skin without the delay of accurately measuring the amount, the syringe being loaded and at once emptied into the subcutaneous cellular tissue.

Sulphuric ether is another prompt and powerful stimulant which may be injected under the skin in the same manner and under the same indications as ammonia. By mouth and in ice water it is also efficient, but is unpleasant and much slower in action than hypodermically. Its effect, too, is transient. *Compound spirits of lavender* and the *tincture of valerian* or *elixir of valerian and ammonia,* are likewise cardiac stimulants of recognized though varying power. The lavender preparation probably owes much of its action to the contained alcohol, while the valerian preparations are intensely disagreeable on account of their odor and taste. They are less eligible, therefore, than are the preparations of ammonia. I have tried repeatedly the combinations of these several stimulants recommended by some physicians for times of emergency and formerly indorsed them myself as powerful stimulants to weak hearts in pneumonia. But the objectionable effect on the stomach has caused me to abandon them except for brief periods, and then singly.

Alcohol.—That this agent is regarded as a cardiac stimulant is proven by the frequency and liberality of its exhibition in the heart-weakness of acute pneumonia. Next to strychnine there is perhaps no one remedy so universally ordered in this emergency. There arises a grave doubt, however, as to the wisdom of such medication growing out of the physiological action of alcohol. As already pointed out in these pages the cardiac incompetence of fibrinous pneumonia is associated with an abnormal lowness of pulse-tension which, referable in the first instance to toxic paresis of the vaso-motor centers, at length becomes accentuated by the failing propulsive power on the part of the heart.

Alcohol primarily invigorates the pulse and increases its volume but does so by causing vaso-motor dilatation, so that there is good ground for believing that the greater frequency and strength of the heart's action is in reality secondary to the vascular relaxation. If now the dose of alcohol be augmented a condition of vaso-dilatation amounting to actual capillary paresis may result. In other words, the action of the alcohol becomes analogous to that of the infection.

Under such circumstances is alcohol a safe remedy in the threatening heart-failure of acute pneumonia? By some clinicians this query is answered in the negative, while by others the reply is in the affirmative. How then is one to reconcile these diametrically opposite views? Only, I think, by the assumption that in some cases the heart-weakness of pneumonia is due to toxic enfeeblement of the myocardium while in others it is mainly the indirect result of capillary paresis. If this be correct, then we have an indication or not for the exhibition of alcohol in the state of the vascular system and, in fact, such appears to be the case.

If, in any given case of acute pneumonia, cyanosis of a more than usually pronounced type precedes the acceleration and enfeeblement of the pulse, alcohol is theoretically contra-indicated. On the other hand, if the pulse begins to grow in frequency and the heart-sounds to become weak, short and perhaps impure, while cyanosis is not very widespread and intense and *pulse-tension has not yet fallen to a noticeably low ebb,* then the judicious use of alcohol may do good and is indicated in connection with the stimulants previously discussed.

Even in this latter class of cases there is great difference in the alcoholic preparation to be selected. The stronger liquors, in particular whisky, rum and gin, are to be avoided if possible and preference is to be given to the wines which contain a relatively low percentage of alcohol and a high percentage of ethers since these latter are highly stimulating and do not act deleteriously as does the alcohol. Hungarian Tokay, champagne and even Burgundy and claret are serviceable, particularly the Tokay. French brandy is often prescribed but, although preferable to whisky, is not equal to some fine wine. Whatever the preparation selected, the amount administered should be small and repeated only as indicated. Should heart-weakness grow, despite the wine, and cyanosis deepen, nothing is to be gained by increasing the dose and frequency of the alcoholic stimulant.

Nitroglycerin.—This is still another remedy that is resorted to in these cases without due regard to its physiological action, and hence the likelihood of its doing more harm than good. This agent is *par excellence* a vaso-dilator and increases the frequency and temporarily the force of the pulse through its action on the vascular system in the same manner as does alcohol. In my opinion its influence upon the heart is wholly offset by its action on the vessels, and hence I believe it is *not admissible in pneumonia,* especially when cyanosis is pronounced. It is advised by some when pulse-tension is high, but as it is my conviction that blood-pressure tends to fall steadily throughout the course of pneumonia I fail to see the force of such advice.

Oxygen.—This is another of the stand-bys in this stage of pneumonia and remarkable claims have been made for the benefit of its continuous inhalation.

For my part I have never been able to convince myself of its possessing any real value except in *cases marked by accumulation of secretions in the air-tubes* and consequent mechanical interference with hæmatosis.

True, cyanosis from capillary paralysis clears up more or less after the administration of oxygen gas and thus it appears to benefit the sufferer. But, to my notion, the improvement is fallacious and in reality speaks for nothing more than a temporary exchange of CO_2 for oxygen in the tissues. It does not in anywise prove that the paresis of the capillaries is lessened.

The heart-muscle may be supplied for the nonce with oxygenated blood and thus may be somewhat stimulated, and herein may lie enough beneficial action to warrant the employment of the gas. It is an adjunct to other more powerful means of stimulation which in so desperate a fight cannot be ignored or spared. Nevertheless, in an abundant supply of pure fresh air, and in particular cool pure air, we possess a means of cardiac stimulation fully equal to the inhalation of oxygen.

Asafœtida.—Doubtless most readers will be surprised by the mention of this agent as a cardiac stimulant. Nevertheless, asafœtida is a powerful stimulator of the nervous and circulatory systems. Previous to my perusal of Fraenkel's recommendation of this remedy in the collapse of acute fibrinous pneumonia I had employed it with unmistakable advantage in the tedious convalescence from pneumonia and from influenza.

The German authority just cited highly recommends asafœtida in doses of 75 grains in an emulsion of 3 ounces of yolk of egg, as an enema in the state of collapse characterized by cold, perspiring skin, feeble, frequent pulse and pinched, anxious countenance. He says such an enema may be repeated as occasion demands.

Acting on this advice such an enema was once prescribed by my interne, Dr. M. W. Hall, to a patient presenting this condition at Cook County Hospital. The effect was truly surprising and was afterwards maintained by hot applications and hot stimulating drinks. After a pseudocrisis this man ultimately succumbed with signs of broncho-pneumonia of the opposite lung. I certainly recommend a trial of asafœtida under similar circumstances. Given by mouth in daily doses of 60 grains it may also prove a useful stimulant to patients showing a tendency to coldness of the surface and exhaustion.

Suprarenal Extract.—In the form of a 1 to 1,000 solution of adrenalin, this agent has been a good deal employed on the theory of its acting as a vaso-constrictor and thus tending to neutralize the vaso-paralyzing effect of the infection. It is particularly recommended in cases showing marked and progressive cyanosis. The dose is 5 minims injected under the skin every two to four hours according to the urgency of the case.

I formerly employed adrenalin to a considerable extent and, on purely theoretic grounds, recommended it in my writings. Upon further experience, however, I have discarded it from my armamentarium because I was unable to satisfy myself of any benefit accruing therefrom. I have been credibly informed, moreover, by a medical man who could have no inducement to make false representations in the matter, that at a certain well-known pharmaceutical laboratory he witnessed experimental injections of adrenaline in 15-minim doses under the skin of dogs with the result that almost this entire amount could be recovered two hours later from the blanched and hardened area marking the site of the injections.

Whether such experiments prove anything or not, they seem to my mind to accord with what might be expected from the well-known styptic action of this drug. Therefore I am doubly skeptical regarding the benefit that may accrue to pneumonia patients from its hypodermic administration. Moreover, as previously stated in this work, Meltzer and other experimenters have seen sudden death follow the injection of adrenaline, presumably because of its constrictor effect upon the coronary arteries. Its use therefore is not without risk.

Venesection.—The reaction that succeeded the extravagant bloodletting of our forefathers, and led many to the abandonment of even leeches and wet cups, appears now in turn to be giving way to a more rational view of the measure. Bleeding is now coming to be looked upon as a highly useful procedure in some diseases which have led to dangerous engorgement of the right heart and venous system. It relieves stasis and gives the heart a chance of adequately driving the blood onward. Accordingly, *when, in acute pneumonia, the right ventricle becomes overdilated and the organ begins to falter in its struggle, venesection is indicated and may prove a highly salutary measure.*

Bloodletting is also indicated when the increasing or widespread cyanosis betokens capillary paresis with its consequent peril. The circulation is tending to stagnation and when blood is drawn from a vein it is often so dark and thick as to flow with difficulty.

Under such circumstances a vein of the arm should be promptly opened, under strict antiseptic precautions, and the blood should be allowed to flow until it loses its grumous character and presents its normal color and fluidity. Even though it necessitate the abstraction of 30 ounces, I believe the benefit resulting justifies the withdrawal of such an amount.

The beneficial effects in some cases are shown by the increased force and volume of the pulse and the greater clearness and intensity of the heart-sounds, in particular of the pulmonic second. It is stated also that the patient may fall asleep, his temperature lower and the crisis occur not many hours thereafter, suggesting the possibility of its having been hastened by the withdrawal of toxic substances along with the blood.

The best method of performing venesection is to cut down on the vein, then pass a director beneath it, and while the vessel is held steady by a pair of forceps to make an oblique slit through its coats. It is well to pass ligatures loosely about the vein before opening it. The flow can then be instantly stopped by tightening the ligatures. Opened in this manner, the edges of the wound can be held apart to insure a free flow if necessary, whereas if the vein be divided, as I once saw done, the severed ends collapse and shut off the escape of blood. When sufficient blood has been taken the wound may be closed by a stitch or two and the arm dressed with antiseptic gauze.

I have known remarkable improvement to follow the letting of 30 ounces. In one instance the man who previously was deeply cyanosed and breathing with great difficulty grew rosy in appearance and expressed himself as greatly relieved. The pulse previously small and feeble became full and strong, and everyone in the family commented on the manifest change for the better. Unfortunately, however, the improvement was not maintained and death occurred a day subsequently. Unquestionably, however, the venesection prolonged, although it could not save, life in this case.

Bloodletting is a justifiable procedure which should be performed before the incompetence of the myocardium reaches an extreme degree. But unfortunately it is usually resorted to only after all other measures have failed and the case is thought hopeless. Therefore, I would earnestly recommend that venesection be decided on, together with redoubled vigor of stimulation, before it is too late, and when there is still a chance of saving the patient.

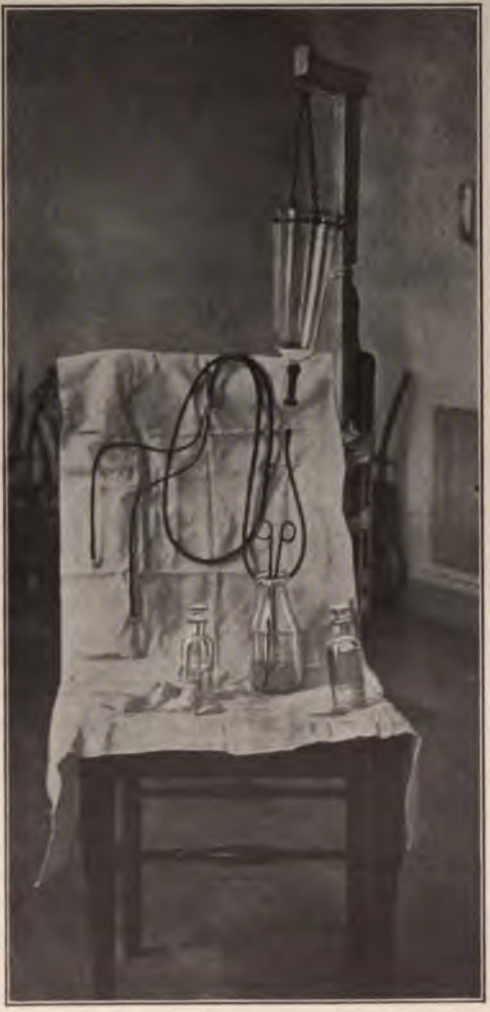


FIG. 32.—Transfusion set.

The Intravenous or Hypodermic Injection of Physiological Salt Solution.—This measure first carried into effect by a Philadelphia physician in 1890 was proposed for the purpose of diluting the blood and at the same time raising blood-pressure in the renal arteries so as to promote diuresis and hasten the elimination of toxins through this natural channel of excretion. The suggestion seems an excellent one and, taken in conjunction with venesection, appeals to the thoughtful practitioner as rational.

The physiological salt solution must be introduced in an amount equal to or slightly in excess of that taken by venesection. It may be injected directly into a vein or under the skin (hypodermoclysis). The apparatus necessary for subcutaneous introduction of the solution may be easily constructed out of a fountain syringe and a fair-sized aspirating needle. It is well to have a short glass tube connect the tube of the syringe with that into which the needle is inserted, that the operator may be able to observe the flow.

If it is desired to obtain a more rapid injection into the subcutaneous cellular tissue than is possible through a single needle, then two of them may be connected with the main tube by means of a Y-shaped glass tube (Fig. 32). The syringe should be suspended about four feet above the level of the patient's body. The solution must be of the temperature of the body and the injection must be made into the loose areolar tissue of the abdomen or inframammary region, under perfect asepsis that suppuration may not ensue.

Hypodermoclysis is painful, but less in the parts named than elsewhere, and a considerable tumefaction generally remains for some hours after the injection. The discomfort is insignificant, on the whole, and not to be considered in comparison with the possible benefits.

Acute pulmonary œdema is a symptom or a condition from which I doubt the possibility of rescuing a pneumonia sufferer. Nevertheless, some attempt in this direction must be made. If it happen that this œdema occur suddenly and unexpectedly in a patient who appeared to be doing well and hence was not receiving stimulation by the means recommended, then, of course, no time should be lost in resorting to the hypodermic administration of camphor, ammonia, ether, musk or brandy in the hope of whipping up the flagging heart. The subcutaneous injection of $\frac{1}{50}$ to $\frac{1}{25}$ of sulphate of atropine will promptly cause a cessation of the râles of pulmonary œdema and a temporary improvement of the patient's state. But whether it can actually save life after this condition has once set in I cannot say. In one case which I was attending together with Dr. G. W. Webster, of Chicago, and which has been narrated in these pages, atropine was administered a number of times and in each instance occasioned a speedy disappearance of the local signs of œdema, but did not save the patient.

It is not very uncommon for a patient to become too weak to cough effectively and the lungs become filled with a multitude of bubbling râles; or tracheal râles alone are heard. In other instances these râles are an evidence of growing feebleness of the right heart. This state of things is not pulmonary œdema but closely resembles it and calls for treatment. Stimulants are now indicated or, if already in use, must be increased. To these may be added the internal administration of squills, camphor or a decoction of senega (Fraenkel) of the strength of 10 to 150.

Alkaline Treatment.—It is altogether premature to make definite statements concerning the efficacy of attempts by means of large doses of sodium bicarbonate to counteract the acidity of the blood which appears to be occasioned by the pneumococcus and to which, possibly, some of the late symptoms may be attributed. Nevertheless, it may not be amiss to mention the fact that, under Dr. Frank Billings's recommendation, a considerable number of pneumonia patients in the Presbyterian Hospital at Chicago have been treated in this manner, and with what appeared to be encouraging results. Herein lies the suggestion, at all events, that the results claimed for salicylate of soda and enormous daily doses of iodide of potash may be due to the ingestion of an alkali in large amount.

Continuous Rectal Irrigation with a Physiological Salt Solution.—With a view to the possible removal from the blood of toxins through increased diuresis this plan of treatment was essayed in the wards of Cook County Hospital during the Winter of 1904–1905. The results have not been tabulated as yet and hence positive statements cannot be made. Nevertheless, I think I can say that the impression was gained that in the cases in which this treatment was thoroughly carried out the effect was encouraging.

The method was as follows: Normal salt solution of the temperature of the body was put into a reservoir (a large fountain syringe) and the bag was suspended on the head of the bed or just above, and the contents were allowed to trickle drop by drop through a rectal tube into the large bowel. Care had to be exercised that the fluid did not enter more rapidly than it could be retained, absorbed and eliminated by the kidneys. Otherwise the return from the rectum caused wetting of the bed. A nurse kept the syringe replenished with fresh solution of proper temperature. No serious complaint of discomfort was entered by the patients thus treated, provided wetting of the bed did not occur. It certainly seemed in a number of instances to lessen symptoms of infection and some patients recovered who, on general principles or because of their condition on admission, were thought to afford an unfavorable prognosis.

The deceptiveness of acute pneumonia is such that, as every practitioner of experience knows, one individual will get well when everything seems against him, while another with everything in his favor, apparently, will suddenly grow worse and die. Moreover, some epidemics appear to be mild, so that the same class of persons will yield very different mortality figures at one season from those at another. It is these considerations that render of absolutely no value reports based on a small number of cases. Therefore I am unwilling to make any claims for this or any other plan of management herein described. I will say only that this continuous rectal irrigation furnishes one more possible means of overcoming the infection of acute pneumonia and hence of sustaining the heart.

Treatment of Complications.—Against most of these we are powerless except in so far as we can maintain the fighting strength of the individual. Thus, endocarditis, pericarditis, meningitis are beyond our powers of control, and the most we can do is to provide good nursing and protect the patient by retention in bed against still further injury until the activity of the process is over and the person is able to resume his wonted mode of life. Many of such patients will die in spite of us.

Empyema, arthritis, suppuration and inflammation of the middle ear must be treated surgically. Abscess and gangrene of the lung, when they do not speedily end in death and when accessible to the surgeon's skill, must also be operated upon and may in this manner be brought to a successful termination.

The method suggested by Fochier and indorsed by Lépint and Dieulafo of attempting to prevent abscess of the lung by *abcès de fixation*, that is, the production of a subcutaneous abscess in other parts, is not known to me by personal experience and does not appeal to me. In the first place the diagnostic signs by which a threatening pulmonary abscess may be recognized are doubtful and unreliable. In the second place the subcutaneous injection of oil of turpentine is very painful. In the third place the effects of such injections cannot always be confined or limited and extensive phlegmon may result. In the fourth place the theory on which this treatment is founded is too uncertain of practical results.

CHAPTER XVIII

ACUTE BRONCHO-PNEUMONIA

THIS variety of pulmonary inflammation is distinguished from the fibrinous form previously considered by its limitation to a group of lobules and by the character of the inflammatory products as well as by its ætiological and clinical characteristics, as will be set forth.

Synonyms.—These are various and have arisen from the desire for a term that would at once indicate the main anatomical differences between this and the primary fibrinous or croupous variety.

Lobular pneumonia is the term given by many authors (Liebermeister *et al.*) to denote the restriction of the process to groups of lobules rather than the involvement of an entire lobe.

Catarrhal pneumonia is the designation preferred by older writers (Jürgensen, Aufrecht) and is selected because the inflammatory products resemble those of an acute catarrhal inflammation of mucous membranes rather than the fibrinous exudate of acute croupous pneumonia.

Capillary bronchitis, which at one time was differentiated from broncho-pneumonia, is now held synonymous with the latter because in catarrhal inflammation of the terminal bronchioles the adjacent air-vesicles are also involved.

Ætiology. **Predisposing Causes.**—*Infancy* and *old age* may be stated to be the most important of the conditions predisposing to broncho-pneumonia. This predisposition of the two extremes of life is intimately connected with the liability at these ages to certain other acute and chronic diseases. Nevertheless, the consideration that healthy adults are usually exempt from this form of pneumonia, even when exposed to the same acute or chronic diseases as predispose infants and old people to broncho-pneumonia, renders reasonable the inference that in infancy and old age, as such, lies an important predisposing factor.

Broncho-pneumonia is especially frequent during the period of the first dentition and even up to the age of five years. Thus Holt states that of the
onias affecting 370 children under five, this form constituted 75 per
d of 72 cases tabulated by Steffan 52 occurred prior to the fourth
lson Fox). As may be expected, the proneness to the complaint
ith the advancing age of children, a circumstance that may probably
ed on the ground of their increasing vigor.

rs recognize and emphasize the proneness of young children to
monia, but that the reader may not gain the impression that

this variety is the more frequent in children of all ages, I may cite von Ziemssen's figures as given by Fox. Of 284 cases of pneumonia in childhood there were 186 of lobar and only 98 of broncho-pneumonia. In this connection it may also be stated that undoubtedly many of the lobular or patchy pneumonias seen in young children belong to the fibrinous variety and are lobular in distribution, but not in the nature of the inflammatory exudate.

Persons of advanced age are also more liable to broncho-pneumonia than are those in the middle period of life but not so frequently as are young children. In this connection it is well to call attention to the fact that genuine fibrinous pneumonia may, and often does, show a lobular distribution. For this reason I believe that many fatal cases of so-called broncho-pneumonia in old people are in reality instances of the primary croupous form, the same as in children. This accords with the statement made by Osler and Delafield that broncho-pneumonia may be primary and may be caused by the diplococcus of Fraenkel. Nevertheless, it is in most instances a secondary affection. This consideration often leads to the belief that cases of pneumonia in the aged with patchy signs and a preceding bronchial catarrh are instances of catarrhal pneumonia when, in reality, they are not.

Bronchitis.—In the old as well as in others of enfeebled resistance this, either acute or chronic, is an important predisposing factor. In such cases the broncho-pneumonia is often stated to be the result of direct extension of inflammation along the walls of the bronchi. It is rather because in the bronchitis are furnished conditions favoring the growth and dissemination of bacteria.

When, by reason of inflammation, the bronchial secretions are increased in amount, altered in character and often are not expelled by coughing efforts, they provide an excellent culture medium for germs that may have been previously present or may have subsequently gained access. If, now, in consequence of violent expiratory efforts attending cough, or of the forced inspiration which precedes the act of coughing, these bronchial secretions with their growing germs are driven still deeper into the terminal ramifications of the bronchial system, there would thus be set up conditions favorable to catarrhal pneumonia.

A secondary or subordinate factor in cases of bronchitis, in particular capillary bronchitis, is the atelectasis so often a result of the blocking of the smallest tubes by secretions or tumefaction of the mucous membrane. If germs gain access to collapsed alveoli their growth and multiplication are insured and inflammation of the alveolar walls can scarcely fail to follow. The foregoing considerations make it plain why *capillary bronchitis* is so dangerous a process in young children, and why it is practically synonymous with catarrhal pneumonia.

The intimate relationship existing between lobular pneumonia and bronchitis suggests that a remote predisposing influence may be attributed to *climatic conditions.* Such as a matter of fact is the case, since this form of pneumonia, like the fibrinous, is more prevalent in cold seasons or whenever the weather, previously warm, suddenly changes and becomes cold and windy. It is therefore most common in the Winter and Spring months.

Acute infectious diseases, viz., measles, pertussis, diphtheria, influenza, variola and, to a less degree, scarlatina, typhoid fever and erysipelas, must be reckoned as among the most powerful and, indeed, the most frequent conditions predisposing to broncho-pneumonia.

Measles is especially likely to set up this form of pneumonia in young children. This is recognized by all physicians of wide experience in infantile diseases and is well shown by the figures cited by Fraenkel. Of 573 cases of measles observed by Bartels, 68 or 11.9 per cent were complicated with broncho-pneumonia. Of von Ziemssen's and Krabler's 311 cases, 50 or 16.1 per cent had broncho-pneumonia. Of Emden's 461 cases 27 or 5.9 per cent showed this complication. The relation of broncho-pneumonia to measles at different ages in childhood is shown by Bartel's figures given by Aufrecht, viz., below one year 19.33 per cent of pneumonia, one to five years 12 per cent, and five to ten years 10.33 per cent.

The unmistakable influence of measles over the development of broncho-pneumonia, as well as the fact that its malign influence is not of necessity limited to childhood, is illustrated by Bartel's case. This was in a woman of forty-six who was destitute and uncared for and who succumbed to broncho-pneumonia. In her case the influence of the acute infection (measles) was enhanced by the unfavorableness of her environment which, as will subsequently be shown, is a highly important factor.

Whooping-cough is also very liable to be complicated by broncho-pneumonia but not so often as is measles. It is the associated bronchial catarrh and the atelectasis which lay the little patients with this complaint open to the danger of pneumonia. According to Fraenkel it is especially so in the convulsive stage. Figures given by Fox show that of 52 cases of broncho-pneumonia observed by Steffan and 98 by von Ziemssen 8 and 23 respectively occurred in pertussis.

Diphtheria also predisposes to broncho-pneumonia, but less commonly even than whooping-cough (Aufrecht). I cannot find in works at my command statistics giving its relative frequency but Fox states that Peter reported 67 cases of "confirmed pneumonia" in 100 of diphtheria, a percentage which Fox regards as too high, while Fraenkel quotes A. Barginsky as having found catarrhal pneumonia in 50 per cent of his fatal cases of diphtheria. This statement will prove surprising to most readers, I think, as it has to me.

The development of lobular pneumonia in diphtheria is generally put down as due to extension, but Fraenkel agrees with Henoch in the opinion that the pneumonia is far more probably a result of the aspiration of infectious material from the throat or trachea.

Influenza is an important predisposing infection but the frequency with which broncho-pneumonia occurs varies in different epidemics and according to the sources whence statistics are gathered. Thus, quoting from Fraenkel again, of 34,556 cases of influenza in the German army there were but 219 instances of pneumonia, i. e., 0.6 per cent, while in the hospitals of Urban and Friedrichshain at Berlin and in the Bürger Hospital at Cologne the frequency ranged from 22 to 24 per cent. Litten gave 6 to 8 per cent as the average taken from Leyden's and Guttmann's statistics for the epidemic of the Winter of 1889 and 1890. From the foregoing it would appear that not only does the frequency differ in different epidemics of influenza, but that the prevalence of *grippe-pneumonia* is largely determined by conditions of environment and other depressing circumstances.

In the remaining acute infections mentioned above the liability to pneumonic complication is not great, but is yet sufficient to have attracted attention and should make physicians in charge of such disorders watchful for its recognition. The precise mode of production of catarrhal pneumonia in these affections is not quite clear. In erysipelas, e. g., it would seem as if the infective agent might reach the lungs by way of the blood or lymph vessels which is one of the ways mentioned by Fraenkel as possible. In typhoid fever, on the other hand, the acute bronchitis so often present renders such a hypothesis unnecessary. It is perhaps not always of importance to be able to ascertain the exact *modus operandi* since we would not be any the more able to prevent the pneumonia.

Gastro-intestinal disorders, particularly in infants, must also be classed among the predisposing causes of this variety of pneumonia. By some clinicians, as Renard, Sevestre, Czerny and Moser, the view is entertained that the mode of invasion, as regards the lungs, is by way of the circulation, toxins being absorbed from the intestinal canal. Others, in particular Finkelstein and Spiegelberg, hold to the opinion that in consequence of the prostration attending severe gastro-intestinal disorders it is impossible for the little sufferers to cough out any particles of food or secretions which may be aspirated into the bronchi during spells of vomiting.

This view seems far more reasonable and is strengthened by the following consideration: Gastro-intestinal disorders in young infants are attended by great prostration, and this state leads to corresponding enfeeblement of respiration and circulation. Even should atelectasis of some portions of the lungs not actually occur, the respiratory and circulatory feebleness would favor the growth of germs that might get into the smaller bronchi. Bronchitis and broncho-pneumonia would be the natural result. Fraenkel appears to coincide with this aspiration theory of the causation of these cases, since he says that infection of the lungs from the bowels by way of the blood is not likely except, possibly, in cases of streptococcus enteritis described by Escherich. To my mind also, such a view seems far more likely than does that of the action of toxins.

Chronic Diseases.—There are certain chronic affections of the lungs and heart which may be included among the predisposing causes of this form of pneumonia. Chronic bronchitis has been mentioned already as particularly powerful in old people. It is a predisposing factor, however, in all such as have become reduced in strength and are unable to cough and expectorate effectively. It is an important factor in young children, therefore, since it favors the collapse of groups of alveoli.

Chronic heart-disease is reckoned among predisposing causes, and largely because stasis within the pulmonary and bronchial vessels tends to the production of chronic bronchial catarrh and because individuals with cardiac disease are often much reduced in strength and vital resistance. It may safely be said, however, that cardiopaths are more liable to secondary croupous pneumonia than to the form now under consideration.

Rachitis may also be regarded as a contributing factor to broncho-pneumonia. This is because, in this country at all events, rickets is seen most often in infants or very young children and in them leads to great debility and catarrhal disorders. Under such circumstances are operative all the forces that have been considered at some length already.

Tuberculosis is likewise included among predisposing causes by some authors, but as the broncho-pneumonia of tuberculous cases is a specific affection it does not properly belong in this chapter. In some of these instances the pneumonia is the result of aspiration of bronchial secretions or of the contents of a cavity, while now and then it follows directly upon the discharge into a bronchus of a caseous bronchial gland. Broncho-pneumonia of tuberculous origin will be discussed in detail in the chapter devoted to pulmonary tuberculosis.

Extensive Burns.—Broncho-pneumonia is a very frequent cause of death in persons who have been extensively burned or scalded. Why this is cannot be definitely stated, but it has been suggested that the catarrhal pneumonia may be due to the action of toxins generated by the great destruction of integument. In some instances, no doubt, the pulmonary inflammation results from inhalation of the hot air or smoke or is a true aspiration pneumonia consequent upon trauma of the upper respiratory passages.

Smoke.—The inhalation of smoke is regarded by some authors as a cause of this form of lobular pneumonia. Aufrecht, in particular, has narrated the instances of three young children, two of whom were aged three and five years respectively, who, having been locked in a room by their mother, a washerwoman, got hold of some matches and set fire to a feather bed. The resulting smoke was very dense and irritating, so that the six-weeks'-old baby died the next day, while the two older children were greatly overcome and unconscious. After their admission to the hospital both developed extensive broncho-pneumonia. The *post-mortem* examination of the younger child showed the pneumonia to be unmistakably of the kind we are now considering.

In the chapter dealing with the ætiology of Fibrinous Pneumonia I have related the case of a seventeen-year-old young man who was admitted to Cook County Hospital shortly after having been rescued from a burning building. Although an autopsy could not be held in this instance the clinical evidence pointed clearly to broncho-pneumonia. There was a widespread intense bronchitis with subsequent development of pronounced dullness and bronchial breathing over the right lower lobe.

It may be questioned whether, in such cases, the pulmonary inflammation is the consequence of the irritating properties of the smoke itself or of injury to the larynx and trachea by the hot air, the local trauma favoring the action of germs already present in the parts, or whether the broncho-pneumonia does not ensue upon the aspiration of tracheal secretions along with bacteria during the state of complete or partial unconsciousness in which the individuals are found. This last view is held by many authors, as Aufrecht, who regards such pneumonias as examples of aspiration pneumonia.

Ether.—Among the various substances, the inhalation of which is sometimes followed by broncho-pneumonia, must be mentioned ether. Such an inflammation of the lungs is spoken of as *ether pneumonia* or *post-operative pneumonia.* Much interest and importance attaches to this form of lung-inflammation both from a pathological and an ætiological standpoint. It is, moreover, apt to prove very fatal. As regards its morbid anatomy it may be classed among catarrhal, i. e., broncho-pneumonias, and is so regarded by such observers as Fraenkel and Aufrecht. The latter, in particular, devotes much wearisome discussion to this phase of the question.

The *modus operandi* of ether anæsthesia in the production of broncho-pneumonia is generally explained on the ground of the irritation of the respiratory passages occasioned by the gas. Such a view seems the more reasonable since ether pneumonia is especially observed after prolonged anæsthesia. On the other hand, Bruns is said to declare that cases of fatal bronchitis and broncho-pneumonia are only observed when the ether is impure, and that if a perfectly purified preparation is administered it occasions only a transient and harmless irritation.

As insisted on by Aufrecht the anæsthetic produces copious secretion of mucus in the throat and trachea as shown by the loud tracheal râles. In many instances, moreover, vomiting is induced by the gas. If, under such circumstances, very great caution be not exercised by the anæsthetizer and perhaps in spite of the utmost care on his part, the patient is likely to inhale some of these secretions along with the germs that happen to be in or habitually inhabit the upper air-tract and throat. This view is supported by the experiments of Hoelscher on dogs. He found that if the animal's head was so placed as to permit complete escape of the secretions, pneumonia did not develop.

Post-operative pneumonias are said to occur with greater relative frequency after laparotomies than after any other operations excepting those on the throat or upper air-passages. In the latter class of cases the pulmonary process is a true aspiration pneumonia in consequence of the inhalation of micro-organisms along with secretions, blood or pus. The pneumonia after abdominal operations is not so readily explained, but is probably also an aspiration pneumonia.

If it does not follow ether anæsthesia *per se*, it may, nevertheless, happen that in consequence of the prolonged and continuous recumbency which such patients are sometimes compelled to maintain, together with inability to cough on account of the pain occasioned thereby, germs are aspirated or migrate into the finer ramifications of the bronchial tree. Such is far more reasonable than is the hypothesis of the action of toxins.

Apoplexy and coma are likewise conditions that predispose to the occurrence of broncho-pneumonia. Either through actual paresis, or insensibility of the epiglottis, the protective mechanism of the parts guarding the larynx fails to work, and secretions as well as particles of food are permitted to enter the respiratory tract. The *modus operandi* of the pneumonia in these cases is analogous to that in dogs after section of the vagus. Such a broncho-pneumonia is sometimes spoken of as a *deglutition pneumonia,* but the adjective aspiration is preferable as being more accurate.

Pneumonia of such origin is a not infrequent cause of death in persons suffering from uræmic or diabetic coma or in those stricken with apoplexy. It must not be forgotten, however, that a *terminal pneumonia,* which is properly fibrinous, may also take place in such patients.

Foreign Bodies.—The aspiration of foreign bodies having their origin outside the human organism, as heads of grain, beads, pins, etc., also set up broncho-pneumonia. This has been treated of in the chapter on foreign bodies in the air-tubes and does not require special consideration in this place. It is only necessary to remind the reader that such inflammatory processes in the lungs are very apt to terminate in abscess or gangrene.

Impure Air.—Finally, before ending this portion of the ætiology it may be stated that authors recognize in the foul air of ill-ventilated dwellings as well as in depressing external conditions in general an important predisposing element. This is especially so in the case of children with measles, pertussis, etc., who are confined within badly aired, overheated apartments instead of being allowed under proper conditions to breathe pure invigorating air free from germs. Doubtless, the same thing holds true with reference to individuals with heart disease, bronchitis, or chronic infections of whatever sort. The importance of adequate ventilation should also be borne in mind in the case of old and enfeebled persons who, by reason of their sluggishness of circulation and fear of draughts, are very prone to keep their apartments close and overheated.

Exciting Causes. *Bacteria.*—It goes without saying that the direct or immediate cause of this, as well as of the lobar form of pneumonia is in every instance the action of bacteria. The question of interest is, therefore, whether or not the broncho-pneumonia is the result of any special germ. The response to such query is found in the statement that pathologists agree in ascribing ætiological influence to quite a variety of micro-organisms: diplococci, streptococci, pneumo-bacilli, staphylococci, diphtheria bacilli and influenza bacilli.

I cannot do better than briefly to summarize Fraenkel's statements on this head which, in turn, are based on Dürck's paper wherein is considered the bacteriology of broncho-pneumonia, especially in children. Of 53 lobular pneumonias among adults, Netter found but a single bacterium present in 39, of which the pneumococcus was present in 38.5 per cent, streptococcus in 30.8 per cent, Friedländer's bacillus in 23 per cent and staphylococcus in 7.7 per cent. Of the remaining 14 cases in adults all furnished evidence of mixed infection, pneumococci and streptococci being more frequent than the other two forms and of these the pneumococcus preponderated.

Of 42 cases of broncho-pneumonia among children Netter found a mixed infection in about one half. It is also singular that in these, the streptococcus was relatively more frequent than the diplococcus pneumoniæ. On the other hand Dürck, in 41 of his own cases of broncho-pneumonia in children, found mixed infection in all but 3. Furthermore, the pneumococcus was far more often present than was the streptococcus. In this regard his observations agreed with those of Queissner, Neumann, Banti, and Kreibich. As respects the broncho-pneumonia occurring in connection with diphtheria, it is quite remarkable that the bacillus of Loeffler was the demonstrable cause in every instance. Dürck thus agreed with the discoveries of Flexner, Kutscher, Frosch, and Loeffler, except that some of these investigators found the diphtheria bacillus mixed with other bacteria.

Morbid Anatomy.—Upon opening the chest the affected lung or lungs present a mottled appearance, with areas here and there of a distinctly purplish tint and depressed below the general surface. In immediate proximity to these bluish zones the pleural surface looks cloudy or may show a thin film of inflammatory exudate. To the touch the lung feels more or less nodular but still crepitant, the nodules varying in size from that of a small pea to that of a cherry stone or hazelnut. These solid masses are scattered through the lung or lobe and hence the appellation patchy pneumonia given to the process by some writers (Fraenkel, Samuel West). The nodules are apt to be most numerous in the lower lobe and along the anterior margin.

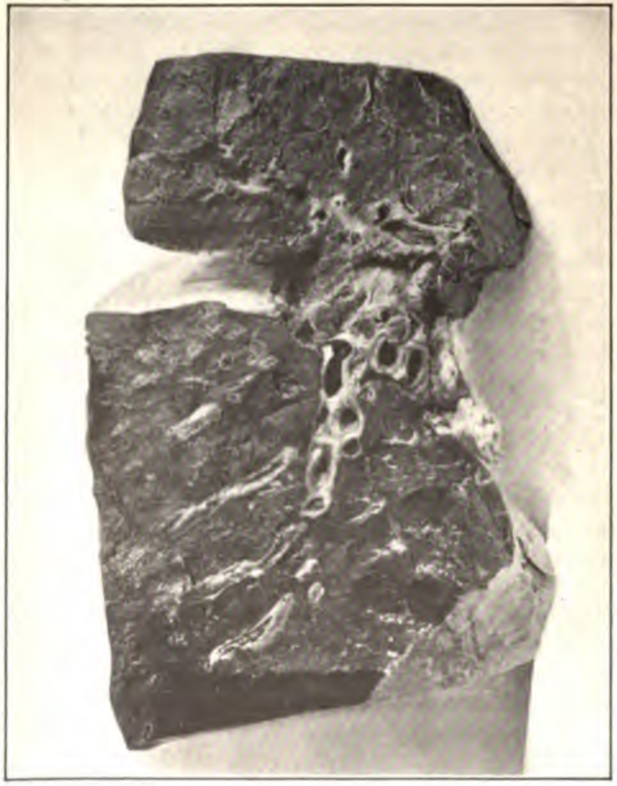
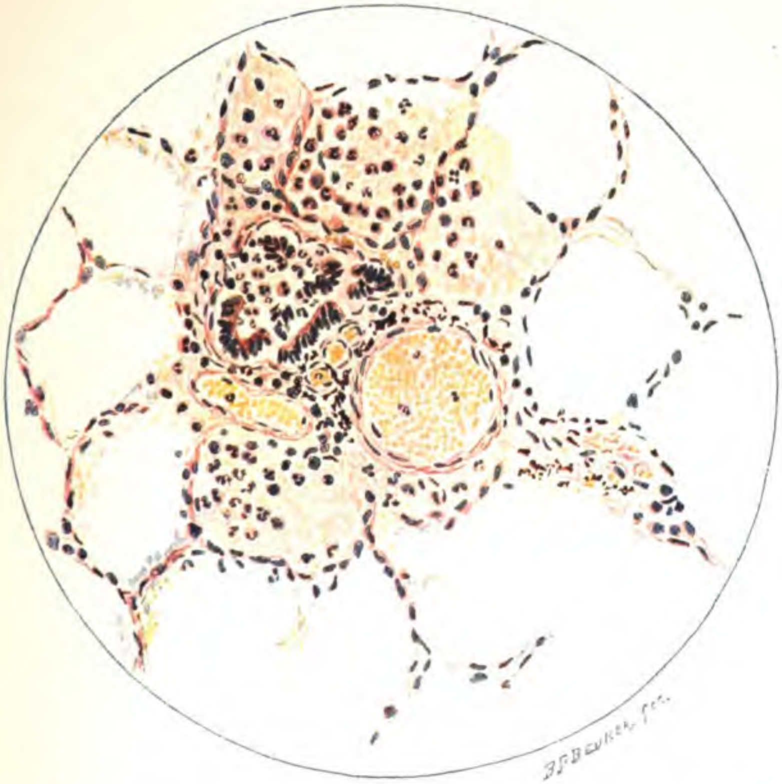


FIG. 33.—Mottled lung of broncho-pneumonia, secondary in this case to epidemic meningitis.

On section the mottled appearance is more distinct, especially when the pneumonic changes are extensive or confluent (Fig. 33). Purplish zones are separated or surrounded by areas or strands of a dark red deeply congested color projecting above the general niveau while here and there are districts of a lighter red hue where the lung is emphysematous. Upon closer inspection small masses or foci of a grayish-red or even yellowish color are seen scattered in the dark red or brownish portions. A bloody fluid exudes from the cut surface which, in most instances, looks smooth or, if granular, much less so than in fibrinous pneumonia.

PLATE VI



Microscopic Section of Lung Showing Early Stage in Development of Broncho-Pneumonia. Bronchus Contains Pus and Desquamated Epithelium, Neighboring Alveoli Invaded by Polynuclear Leucocytes and Serum. (Stained with Hæmatoxylin and Eosin.)

Close examination shows that the dark bluish areas correspond to zones of atelectasis while the firm nodules correspond to lobules or groups of lobules that have become inflamed and hepatized, hence the name lobular pneumonia. If the grayish-yellow foci be subjected to pressure it is found that a small drop of pus can be expressed.

It is only when these broncho-pneumonic areas are closely set or confluent and involve the greater part or whole of a lobe that the appearance of the lung may resemble that of acute lobar pneumonia. Even in such a case a more or less striking difference is presented by the surface of the cut section, inasmuch as it is less granular and sticky and may be even quite smooth. In some cases the resemblance is so close that even pathologists of great experience may be perplexed. In general it may be stated that the consolidation of broncho-pneumonia is less compact and uniform and the transition from red to gray hepatization more abrupt and pronounced.

Microscopically, the terminal bronchioles, infundibular passages and air-cells are seen to be filled with epithelia and leucocytes, the former usually predominating, and hence the name catarrhal pneumonia. Red cells are not numerous and the inflammatory products are less rich in fibrin than in acute croupous pneumonia. There is generally more or less evidence of bronchitis of the finest tubes. The walls of the tubes are often somewhat dilated, this bronchiectasis being apparent when section has been made through their long diameter. When thus exposed the bronchioles look filled with purulent material. The capillaries are congested and encroach upon the diameter of the air-cells (Plate VI).

The alveolar walls are denuded of epithelium, thickened and infiltrated with cells, as are likewise the walls of the bronchioles. This tendency to interstitial thickening is, according to Delafield, the striking characteristic of this form of pneumonia. He says, "Such a broncho-pneumonia differs from the ordinary lobar pneumonia very decidedly; the inflammatory process is not a superficial one, resulting only in filling the bronchi and air spaces with inflammatory products, but it affects also the tissue of the lung, infiltrating the walls of the bronchi and of the air spaces."

Results.—In favorable cases, i. e., of transient duration, this inflammatory process may subside, the cells undergo fatty necrosis and be absorbed, the epithelium lining the air vesicles be regenerated and the lung restored to its former healthy state. In other cases the termination may be very disastrous.

The inflammation may be so violent that leucocytes greatly predominate so that gray hepatization becomes predominant and, under the influence of pyogenic or putrefactive bacteria, localized abscess or gangrene may result.

In chronic cases the interstitial thickening described by Delafield becomes pronounced and limited to small zones, or the greater portion of a lobe may be converted into fibrous tissue. The walls of the blood-vessels do not share in this process of interstitial thickening, however, and hence the lumen of the vessels is undiminished. Occasionally cheesy nodules may be discovered.

Accessory post-mortem findings may be such as have led to the broncho-pneumonia or have resulted therefrom. These are tuberculous or even caseous bronchial nodes, signs of widespread acute or chronic bronchitis, atelectases and dilatation of the right heart.

Symptoms.—Clinically, this affection may be divided into two classes, primary and secondary, according to its manner of invasion. Primary cases set in more or less suddenly, either without signs of antecedent bronchitis, or so closely thereafter as to make it difficult to say which of the two preceded. Secondary cases arise more slowly in the course of bronchitis or some one of the acute infections to which young children are especially liable. In both classes of cases the general features are essentially the same, only, in primary ones, the duration is apt to be shorter and the termination more prompt.

General Clinical Picture in Children.—More or less abruptly as determined by the previous health, a child of tender age, who has an acute bronchitis or is suffering from measles, pertussis, diphtheria or gastro-intestinal catarrh, shows signs of being seriously ill. It is noticed to be listless and feverish and, if the temperature be taken, this is found to be several degrees above normal, 102° F., or even higher. The eyes look dull and heavy, the child is fretful if disturbed and cries when put out of the mother's or nurse's arms.

Cough develops or, if previously present, changes in character, becoming frequent, short, dry and painful as evinced by a little moan or cry. Both respirations and pulse are increased in frequency, especially the former. Thirst may be manifested but desire for food other than liquid nourishment is lost. The tongue is coated and the bowels often disturbed.

If the chest be examined, it is very apt to show hyperresonance of a tympanitic quality in front and at the base behind, also unimpaired resonance, or possibly on one side a rather high-pitched, slightly tympanitic or wooden note. If palpation is made by grasping the back of the chest firmly yet lightly between the outspread hands (see Fig. 27), while the child cries or speaks, pectoral fremitus is very likely to be found distinctly increased over one or more areas in the lower lobes. By auscultation the breath-sounds are perceived to be rapid, short and harsh but not distinctly tubular and accompanied by numerous sibilant or subcrepitant râles.

In some instances these whistling and small-sized crackling râles are so numerous as wholly to obscure the breath-sounds and, no dullness being detected, constitute the sole physical signs on which the physician can rely. They are apt to be more abundant and diffuse at one base than at the other. Exceptionally, there may be distinct dullness in one of the lower lobes behind, which develops by the third or fourth day and over which area, vocal fremitus, bronchophony and bronchial breathing may be marked with creaking or grating sounds suggestive of pleural friction. In such a case the findings are very like those of lobar pneumonia and are probably due to pulmonary collapse rather than to the broncho-pneumonia, although this may occasionally involve a considerable portion of a lobe.

In bad cases the little sufferer grows worse day by day and is plainly losing strength. The breathing is extremely rapid, 60, 80 or more in a minute, very shallow, perhaps irregular and dyspnœa is shown by inspiratory sinking of the soft parts and distention of the nostrils. More or less cyanosis is apparent in face and fingers, and the pulse is frequent, feeble and perhaps irregular. Cough becomes less frequent or is practically suppressed in consequence of the obtunding influence of the carbonic-acid poisoning, and the child is soporose.

Food and medicines are refused partly because of anorexia but more particularly on account of the air-hunger which makes the little patient uneasy and cry as soon as the cup or spoon nears the mouth. The child grows thinner and weaker with each day, the temperature, at first persistently high, 104° or 105° F., becomes irregular and often very erratic, the heart grows more rapid and more feeble with steadily increasing signs of dilatation, and death supervenes after a week or ten days.

In other cases gastro-intestinal and nervous phenomena play a much more important rôle. The onset is with headache or convulsions or with vomiting and diarrhœa. This latter may be a predisposing condition or may be a manifestation of the infectious agent which, in the lungs, produces the pneumonia. Cases in which the cerebral symptoms are marked may be mistaken for meningitis, although West thinks this is less likely than in acute croupous pneumonia. In some cases brain symptoms, i. e., convulsions, do not appear until late and are then of very grave portent.

In *primary* cases that run a more favorable course the clinical appearances are less severe. The pyrexia may be high, but respirations are not so rapid, the heart's action is less disturbed, cyanosis is not so marked and strength is not so rapidly lost. Signs on the part of the lungs are not so widespread and may even be quite limited in extent and consist chiefly of sibilant and subcrepitant râles at the posterior base of one or, it may be, of both lungs. At the end of about a week the general condition improves and the disease ends by crisis or a rather short lysis. Convalescence is, however, apt to be less rapid and uninterrupted than in the true lobar form.

Secondary cases develop in the course of acute or chronic bronchitis, especially in children who are cachectic and hence are most often seen in the overcrowded portions of large cities and in asylums or hospitals for the children of the poor. Secondary broncho-pneumonia is especially prone to occur in the course of the acute exanthemata, and is then often the result of bad hygiene or carelessness on the part of the nurse in allowing the child to become exposed to a draught or a sudden change of temperature. It generally sets in after the appearance of the eruption in measles, but may rarely precede the rash (West), which then appears only after subsidence of the pneumonia.

As remarked by West, the circumstance that the eruption has disappeared prior to the invasion of the pneumonia or that it fades out with the access of high fever is popularly taken to signify that the recession of the rash is responsible for the pulmonary complication. This is an error, since, in the light of modern bacteriological knowledge, we know that an ætiological connection between the disappearance of the eruption and the advent the pneumonia is impossible. Hence there can be small danger from ences thought to "drive in the rash," and measures calculated to "bring the rash out" are futile attempts toward preventing a broncho-pneumonia.

The invasion of the pneumonia in these cases is declared by sudden development of *pyrexia de novo* or exacerbation of such as may have existed, and by the various signs which, in a young child, indicate to the experienced attendant or watchful mother that the little one is ill. They are essentially such as have already been described. The constitutional disturbance is sufficiently pronounced, but in all cases the striking features are those pertaining to the respiratory and circulatory systems.

Cough may be very troublesome but is rarely, if ever, so conspicuous a symptom as is the dyspnœa. This is not complained of in words even by children old enough to describe their sufferings but is shown by the frequency and superficiality of the breathing. Fifty to 60 respirations in a minute are common enough, while small babies may breathe 70, 80 and even 100 times to the minute. Such a respiratory rate is rarely, if ever, absent in cases characterized by capillary bronchitis.

As the disease progresses and oxygenation of the blood grows less and less complete, the condition is very aptly described by the old term *Suffocative Catarrh.* The little sufferer is plainly fighting for air and is in danger of asphyxia, as shown by hurried, panting breathing, corresponding dilatation of the nostrils and depression of the yielding lower portions of the chest. The pulse is also greatly accelerated, so that the disproportion between its rate and that of respiration is rarely so marked as in croupous pneumonia. In infants under twelve months it may readily become too rapid to be accurately counted. In severe and prolonged cases with signs of gradual but inevitable failure of the vital powers the breathing is apt to become irregular, with periods of apnœa, and may assume the typical Cheyne-Stokes character. It is needless to say this is a bad omen, since it portends a fatal issue. The face takes on a dusky hue, looks anxious or, in some instances, apathetic. There may be restlessness or the carbonic-acid intoxication may occasion somnolence and even coma more or less profound. As time goes on the little patient wastes away in strength and flesh until, weak and emaciated, it no longer struggles for breath, coughs feebly if at all and only cries or struggles when disturbed by attendants.

In more favorable cases signs of respiratory and circulatory embarrassment are less pronounced and cough is frequent, hacking and painful. It not infrequently excites vomiting and prompts the child to refuse drink and nourishment. In all cases of broncho-pneumonia loss of strength is progressive and in severe or protracted ones prostration may become extreme. Indeed, in some instances, it is this feature which creates apprehension in the mind of the physician even more than the distinctively pulmonary symptoms.

The clinical features of secondary broncho-pneumonia are well illustrated by a case which I saw with Dr. C. W. Barrett on May 6, 1903. The patient was a large, fat, healthy girl of three years who took ill, on or about April 23d, with what her parents considered merely a cold. On April 25th, as she seemed to be getting worse, Dr. Barrett was summoned. He found her very bright and active but with febrile temperature, about 103° F., and signs of extensive bronchitis. A day or two later he detected a small patch of dullness at the right base behind and her fever ran from 104° to 105° F., while respirations were 50 to 60 and pulse was 120–135.

Dullness spread gradually up the back until it reached from base nearly to apex with bronchial breathing and copious crepitant and subcrepitant râles. This state of things persisted for nearly a week when the temperature fell several degrees and the doctor hoped the worst was over. The next day, however, the temperature again rose to about its former height and signs of pneumonia were discovered at the left lower back. As with the other lung the dullness gradually spread up and down though it did not become so extensive. The breath-sounds were bronchial and there were râles of the same character as at the right.

The child took nourishment well and seemed to hold her strength remarkably well, but pulse and respirations were very rapid, slight cyanosis appeared and at times the little patient showed great restlessness. She did not manifest pain, but cough was frequent and what sputum there was, was swallowed, a very common circumstance in young children, so that its nature could not be determined and it could not be examined microscopically.

Such was the history, in brief, up to the date of my visit twelve days after the attending physician had taken the case in charge. I found a still well-nourished child who was lying partly turned to the right side, quiet but breathing with great rapidity rather than with difficulty, sixty times a minute. She was perfectly conscious and extended her hand in greeting and offered no objection to examination, in this respect being unlike most young children who resist or cry when disturbed by the examining physician. Moderate cyanosis was visible in the face and fingers but there was not marked inspiratory sinking of the soft parts above or below the chest. The abdomen was considerably distended and its upper zone both looked and felt especially prominent, owing, as was found by palpation, to engorgement of liver and spleen. In the young child these organs are very distensible, and whatever interferes with pulmonary circulation occasions early and easily detected enlargement of the liver and spleen.

The skin was hot and dry, the pulse was very rapid, 140, but of good quality, and the heart-sounds were clear. The lungs anteriorly were hyperresonant and the exaggerated respiratory murmur was not accompanied by râles of any moment. At the back, however, there were numerous signs of extensive mischief. On the right, dullness of variable intensity existed from the middle of the scapula to the base, while the upper lobe was distinctly impaired. Bronchial or semi-bronchial breathing was everywhere present while over the lower lobe were abundant moist and dry crackling râles of varying size. A very similar condition prevailed over the left back though dullness was more distinctly patchy. Breath-sounds were not so typically bronchial except in a small area directly below the inferior angle of the scapula. Over this patch the râles were small, dry and crackling while round about were copious moist râles of larger size, bubbling rather than crackling.

difference lies in the ætiology and mode of onset. Catarrhal pneumonia is generally preceded by signs of bronchitis and is more insidious in development.

On examination, the chest is apt to present more numerous and widespread mucous and sibilant râles in both lungs, while dullness, if it can be determined, is far more likely to be in several areas on both sides of the chest. Frequently there is hyperresonance rather than dullness, and breathing is harsh rather than tubular. Sputum is rare, the same as in lobar pneumonia, but if raised is not rusty, being that of bronchitis, mucous or muco-purulent.

Prognosis is most grave since the mortality is high at this as well as the other extreme of life. Recovery may ensue in comparatively vigorous old persons, provided the lung-involvement be not too extensive. Very much depends upon the patient's resistance, however, and, it must be confessed, upon the absence of all depressing measures in the plan of management. It takes such a convalescent a great while to regain former strength and there is great danger of a recurrence.

Influenza Pneumonia.—Thanks to the painstaking investigations of Leichtenstern, Pfeiffer, Wassermann, Beck, Fraenkel, Aufrecht, and others, it has been conclusively established that the pneumonia caused by the influenza bacillus is a broncho-pneumonia. It may have a lobar distribution in some instances, but it has been abundantly proven that this lobar involvement is due to dissemination through the lobe of numerous foci of lobular inflammation, with surrounding districts of splenization. The cut surface is smooth and not granular, and microscopic examination shows infiltration of the alveoli with cells and leucocytes in great numbers in the midst of which, by proper staining methods, can be seen pure cultures of the influenza bacillus.

According to Leichtenstern there may be a purely catarrhal pneumonia or there may be forms showing varying degrees of fibrinous and cellular infiltrations. In how far mixed infections with pneumococci or streptococci are responsible for such mixed forms of pneumonia is not yet definitely established, but the possibility of mixed infection is not denied, is rather admitted by investigators.

Cases of genuine fibrinous pneumonia are confessedly more numerous during *grippe* epidemics and so many of them display deviations from the typical course and symptoms that clinically, at all events, one can hardly escape the conviction that influenza bacilli as well as pneumococci are concerned in their production.

A pneumonia of influenzal origin usually arises after the primary disease has existed for four or five days, but it may develop almost coincidently with the earliest symptoms of the *grippe* or it may set in during convalescence as a result of some imprudence. The symptoms are severe and characterized chiefly by intense depression of the strength, marked irregularity of the temperature curve and a tendency to profuse perspiration.

In the beginning there is apt to be much pain of a dull aching character in the back and the extremities and pleuritic pains are said to be especially intense. The heart's action is rapid and weak and on slight exertion the pulse grows disproportionately feeble, almost thready. Respirations are accelerated and the patient is apt to complain of great oppression of the chest. Cyanosis may become pronounced. Cough is harassing and painful, and the sputum is fluid, somewhat tenacious, of homogeneous consistence and yellow with a faint tint of green. It contains the germs responsible for the affection.

The pulmonary findings are variable and often perplexing. There may be no clearly demonstrable dullness, only innumerable râles of fine bubbling or coarsely subcrepitant character throughout the lungs. Or small scattered areas of more or less defined dullness may be detected over which bronchophony and fine crackling râles exist. In other cases there is well-marked dullness at one or the other apex, or in a lower lobe, or perchance in the middle lobe of the right lung.

Ordinarily the broncho-pneumonic foci are multiple and scattered, making the results of percussion quite different from those of lobar infiltration and sometimes rather uncertain. A peculiarity on which stress is laid by Fraenkel is the changeability of the findings from time to time. At one examination an area of dullness with bronchophony and abundant, sharply consonating râles is detected, and a day or two subsequently this area has vanished and another has made its appearance elsewhere. To what such rapidly shifting changes are owing is difficult to say. They probably depend upon more or less transient areas of congestion or collapse and on the state or amount of bronchial secretions. The heart-tones are rapid and feeble and may be obscured by copious bronchitic râles.

The prognosis is grave, since it is this complication which is largely responsible for the deaths in epidemics of *la grippe*. According to Fraenkel the mortality from this form of catarrhal pneumonia averages 17 per cent and yet, in some of the German hospitals, it has been known to reach 40 per cent. Even if recovery takes place, convalescence is slow and tedious and recurrences are extremely liable to be experienced. Patients remain weak and inclined to unnatural perspirations for several weeks. Cough is apt to be intractable and persistent, and one is often surprised by the detection of fine bubbling râles at one or both bases for a long time after an individual, not quite restored to former health and strength, has yet returned to his ordinary vocation.

Aspiration Pneumonia.—In broncho-pneumonia of this origin the symptoms present considerable variation according to the cause and the virulence of the germs carried into the lungs. In ether pneumonia, e. g., the symptoms may develop soon after the narcosis is ended or not appear until a day or two following the operation. In the former event, as well as in cases of uscitation from drowning, tracheal râles may be noticed directly after the ient regains consciousness and may be audible over the larger bronchi indeed over the back of the chest.

Cough is present and the temperature rises to a variable height. The ations may not be especially accelerated, yet, on examination of the slight dullness may be perceived at one or both bases behind. Breaths are feeble and indeterminate, or perhaps bronchial, and are accom-

These signs were taken to indicate double catarrhal pneumonia with atelectasis and extensive bronchitis. In the light of this diagnosis and of the history, the prognosis was believed to be most unfavorable, very little hope being held out of recovery.

On May 22d I saw the little patient again, and was struck by the great change for the worse. She was much emaciated, lay upon her right side and moaned with evident objection to being disturbed. The pulse was rapid and weak, respirations were from 60 to 70, temperature was persistently in the neighborhood of 101° F., the skin was rough and slightly inclined to moistness, there was an occasional feeble, slightly rattling cough and the breath emitted an odor very suggestive of pulmonary gangrene. I learned that this fœtor had been present for about ten days, and at times had been very intense. It was evident, almost at a glance, that the child could not hold out many days longer, especially as it was difficult to get her to swallow nourishment.

Anteriorly the chest was practically negative but over the back both lungs emitted a dull slightly tympanitic note, especially on the right, while breath-sounds were bronchial and largely obscured by fine and coarse râles that seemed mostly dry and so sharply defined as to be consonating. The voice during her moans was distinct but not intensified or at all cavernous.

Notwithstanding the odor of the breath it was believed that the condition was not one of gangrene but of chronic catarrhal pneumonia with multiple bronchiectases which favored retention and decomposition of the secretions. Death occurred a week or so later, after a duration of five or six weeks.

Symptoms on the side of the genito-urinary system are not common (West) and consist rarely of more than the scantiness and high specific gravity of the urine observed in febrile affections. There may be, however, a trace of albumin and even casts indicative of acute nephritis when the catarrhal pneumonia occurs in the course of some acute infection. Even then the nephritis is due not to the pneumonia, perhaps, so much as to the primary infection.

Pyrexia is a constant accompaniment of this form of pulmonary inflammation, but in type is not uniform, unless in its tendency to irregularity. The temperature is relatively high from the start and for a time may be quite continuous. As days go on, however, its curve tends to become more irregular and may be characterized by sudden and erratic drops and elevations which give it almost a hectic type (see chart, Fig. 34). In protracted and unfavorable cases the temperature tends to be more persistently low, on account, perhaps, of the waning vitality and diminishing resistance.

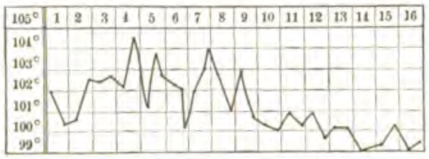


FIG. 34.—Chart of temperature in characteristic case of broncho-pneumonia (Holt).

As a condition of collapse grows more apparent the extremities become cool, the skin is moist or clammy and even the internal temperature is found not to be elevated more than 2° or 3° F. If a case is to terminate in recovery, the character of the body-temperature improves by manifesting a lytical decline until, at length, there may come periods of normal temperature-records with only occasional and transient flurries in which the thermometer may register a rise of a degree or two. As broncho-pneumonia displays a remarkable inclination to relapses, of less severity, than in the first attack, however (West), the temperature chart may show returns or exacerbations of fever running through a series of many weeks or even months and hence the thermometer should be regularly employed as long as the child is not completely restored to health.

The *course* of lobular pneumonia is exceedingly variable. In primary cases recovery may take place within a fortnight or even a week, that is, the pneumonia itself may subside and active symptoms disappear. Convalescence is very slow, however, and there is strong liability to fresh attacks. Unfortunately, the majority of cases do not eventuate in recovery, especially in children below the age of three years, the average of Holt's mortality figures for the first three years of life being 51.33 per cent. Secondary cases are protracted and may drag on over many weeks and even months—"three months and more" (West). Although such prolonged convalescence is extremely tedious and fraught with danger of serious or even fatal complications, still, ultimate restoration to perfect health is possible and the child may grow up to enjoy a life of vigor and usefulness. In other cases with unfavorable environment, the pulmonary changes may be such, viz., fibrosis and bronchiectasis, as render the child sickly and feeble for the balance of his life.

Broncho-pneumonia in the aged does not differ essentially from that of children. It ordinarily attacks old persons enfeebled by bronchitis or the *grippe* and its onset is insidious. Its occurrence is undoubtedly favored by the sluggishness of their circulation and their inability in their weakened state adequately to expel the plugs of tenacious mucus that block the bronchi. As long as the bronchial catarrh is confined to the larger tubes, inflammation of the parenchyma is not likely. But when, from debility and exposure, an acute exacerbation of their catarrh takes place and signs of inflammation of the finer tubes are perceived, danger of catarrhal pneumonia is imminent.

More or less elevation of temperature marks the invasion of the broncho-pneumonia and breathing grows rapid. The pulse increases in frequency and is at the same time feeble, often irregular. Cough is present but is rarely troublesome. In short, the symptoms, although of the same general characters as in children, are less pronounced.

Cyanosis with pallor shows in the countenance, but unless capillary bronchitis is present injection of the cutaneous capillaries is not so marked and
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panied by râles. With the progress of the case these signs grow more distinct and spread over a larger area.

In other instances the indications of a broncho-pneumonia do not appear for eighteen, twenty-four or forty-eight hours and are then essentially the same as just described. Now and then it happens that signs of lung mischief make their appearance for a day or two and then disappear without serious effect on the general condition. In such instances the physician is at a loss to decide whether the signs are really those of broncho-pneumonia or of a localized bronchitis with atelectasis.

Such a case has been narrated to me by Dr. Bayard Holmes, Jr. The lady, previously healthy, with exception of a slight catarrhal cough, endured appendectomy under chloroform, the anæsthesia lasting but fifteen minutes. Toward evening of the following day, some thirty hours after the operation, she developed a frequent nagging cough with slight rise of temperature. Physical examination disclosed an area of dullness of about the size of the palm of the hand, extending from the middle of the right scapula to just below its inferior angle. Over this patch were distant bronchial breathing and fine râles. Heroin allayed the cough, and within the next two or three days the signs of pneumonia wholly vanished. The sputum contained Fraenkel's diplococci.

Unfortunately, not all cases of aspiration pneumonia subside so quickly nor prove comparatively trivial, whether from ether or not. If the *materies morbi* be a foreign body, as a fishbone or button which carries along with it bacteria of an active kind, the symptoms may be violent. Cough is likely to be frequent and severe and there may be dyspnœa. Fever sets in and rises to a considerable height. Signs of localized bronchitis are likely to be present and examination may disclose the seat of the broncho-pneumonia. Such cases are likely, in time, to terminate in abscess or gangrene when, of course, constitutional symptoms become most grave (see chapter on Pulmonary Abscess and Gangrene).

Aufrecht has graphically described the case of broncho-pneumonia following attempted suicide by means of charcoal fumes which may here be given briefly as follows. When the man was transported to the hospital March 4th, he was in a state of extreme prostration, unable to speak above a whisper and very pallid, with small, immobile pupils. His temperature was 100.2° F. A mass of brownish mucus adhered to his lips and cheek. He was able to swallow a little fluid administered to him with great care.

The next morning his condition remained about the same. Pulse was 90 and temperature was 101.7° F. March 6th his temperature had risen to 104° F. and henceforth until his death, March 10th, it ranged between this point and 101.1° F. On the second day after his admission examination detected a small patch of dullness, with feeble bronchial breathing and râles at the right base behind, which gradually spread upward to the spine of the scapula. On the morning of the day he died albuminuria was discovered for the first time. At length, delirium and cyanosis set in and he died six days after his entrance to the hospital. The autopsy verified the diagnosis and revealed ulceration of both vocal chords, which accounted for the patient's inability to speak above a whisper.

Mention has before been made of a case of broncho-pneumonia following the inhalation of smoke observed by me at Cook County Hospital in the Winter of 1905. The symptoms in this case were severe and were characterized by high irregular fever (see Fig. 19), marked rapidity of respiration, prostration and inability to speak above a whisper. The objective physical signs consisted of numerous fine mucous râles throughout both lungs, a patch of dullness with obscure bronchial breathing at the left base behind, and in the right lower lobe a larger area, which gradually spread until the greater part of the lobe presented unmistakable signs of pneumonia. Thirst was a prominent source of complaint and death took place under symptoms of marked cyanosis, restlessness and delirium.

Pyrexia is present in nearly all cases of aspiration pneumonia, but in speedily fatal cases may be wanting (Aufrecht). It is irregular and may show striking differences between *maxima* and *minima.* The respiration is usually embarrassed and rapid, but cough need not be pronounced, and expectoration is not always present or is scanty and mucous or muco-purulent. The signs of consolidation are less pronounced and uniform than in the fibrinous form, the patches often being scattered and small. The course may be mild or severe and in duration the pneumonia may last from a few days to weeks. The termination may be in recovery, abscess, gangrene or death, according to the severity of the case.

Physical Signs. Inspection.—Broncho-pneumonic patients always display more or less evidence of interference with respiration and circulation. Their dyspnœa is shown by rapidity of breathing, inspiratory play of the nostrils and, in the young especially, by inspiratory retraction of the lower portion of the sternum, adjacent costal cartilages and epigastrium. Defective hæmatosis is shown by duskiness of the countenance that may, as the disease progresses, deepen into pronounced cyanosis of face and extremities.

Palpation.—This may be a positive aid to diagnosis in infants and young children since in them the chest-wall is a wonderful conductor of vibration. It implies the necessity of getting the child to utter a sound, and babies may be induced to cry when not so soporose or feeble as to be unable to cry. In all cases, however, palpation may be of aid only when areas of induration are of sufficient size or so located as to conduct vibration to the parietes. Under such circumstances the pectoral fremitus is generally increased.

Percussion.—The value of this means of exploration depends upon the size and number of the broncho-pneumonic foci and whether or not they are masked by emphysema. In most instances there is circumscribed dullness at one or both bases but sometimes the zones of inflammation are too small to yield demonstrable dullness of themselves and it is the associated atelectasis or splenization, or both, which really cause appreciable dullness. *In young children* percussion should always be very *lightly performed,* and when so made may bring out impairment of resonance that would with heavy percussion-strokes be completely lost.

In very many cases in which foci of patchy pneumonia are small and scattered the percussion-note is hyperresonant or tympanitic on account of accompanying emphysema, especially in front along the margins. In fact, only such exaggeration of resonance, without any appreciable dullness, is all that anyone can detect. In infants it is possible for too strong percussion in front over the main bronchi to elicit a veritable cracked-pot resonance. This is especially so when bronchitis exists but, according to Wilson Fox, may be the case even without bronchitis. This should be borne in mind, lest the production of such a cracked-pot note lead one to an erroneous diagnosis by the supposition that cavity exists.

Auscultation.—This is a very important means of diagnosis and is, in many cases, together with inspection, the main dependence in a correct interpretation of the case. Râles are the principal thing perceived by auscultation. These may be both dry and moist, i. e., sibilant and mucous, but in general they are fine bubbling, sharply crackling or consonating. They are very often so numerous, widespread and loud as wholly to obscure the character of the breath-sounds. Whenever the respiratory murmur can be heard, however, it is bronchial or semi-bronchial over the pneumonic foci. Auscultation of the voice-sounds is also highly important, since, according to Leichtenstern, the recognition of bronchophony in disseminated areas furnishes valuable information of the existence of lobular pneumonia.

Complications.—These are not frequent and are not numerous. Acute pleuritis may be occasionally observed when the pneumonic area lies directly beneath the visceral pleura, but seldom, if ever, materially affects the clinical picture or course of the primary affection. Nevertheless, empyema may complicate measles as was observed in a single instance by Aufrecht. As an additional complication of broncho-pneumonia in the course of measles may be mentioned pericarditis, which was discovered *post mortem* by Wyss. This author also recognized a pericardial rub in a twenty weeks' old infant who suffered from catarrhal pneumonia secondary to bronchitis.

Miliary tuberculosis as a complication of this form of pneumonia is variously regarded by authors. Thus Wyss is said to consider it frequent (Aufrecht), the tubercles being found in the pulmonary tissue immediately surrounding the pneumonic focus, or in the meninges of the brain. Von Ziemssen, on the contrary, regards acute miliary tuberculosis as a rare complication, agreeing with Bartels who found but two instances in twenty-one autopsies on cases of catarrhal pneumonia.

Aufrecht holds the same view regarding its frequency and thinks the contrary opinion is based on erroneous interpretation of clinical and pathological findings. It is very possible that in many instances the broncho-pneumonia is a consequence and not a cause of the tuberculosis.

Terminations.—There may be complete or partial recovery, depending on the nature of the cause and various other factors to be set forth in the prognosis; or there may be pulmonary abscess or gangrene or death. In young children a broncho-pneumonia is likely to prove fatal, but in some instances it merges by degrees into the subacute or chronic form from which the patient at length recovers.

Abscess or gangrene, when it occurs, does so most often in an aspiration or deglutition pneumonia, as has already been stated. Such a result may very rarely be observed in the catarrhal pneumonia of measles (Wyss and Damaschino). The abscesses observed by the latter and declared to be extremely infrequent are said to be of the size of a nut.

The transformation of the broncho-pneumonic area into fibrous tissue, i. e., a cirrhosis or chronic interstitial pneumonia, is said to occur in cases which pursue a very chronic course. Such a process can only affect the subsequent health of the individual when it is of considerable extent.

Diagnosis.—The recognition of the presence of catarrhal pneumonia depends in many instances upon other factors than the results of chest-examination. These are history, mode of invasion, symptoms, course and termination; and consequently a positive diagnosis early in the malady is not always possible. In such cases one must content himself with an inferential diagnosis.

History.—The disease in question is most common in young children, e. g., below the age of three years, next in old persons, and is rare in robust young adults. There is generally a history of antecedent bronchitis which is either idiopathic or more often dependent upon some acute infection, as measles, pertussis, influenza, diphtheria, typhoid fever, etc. Or there may be a history of the aspiration of a foreign body or of some disease, operation or other condition predisposing to the inhalation of germ-bearing particles into the bronchi.

The *invasion* is not sudden as in the fibrinous form, but is insidious and takes place during the existence of some predisposing affection. The sudden elevation of the temperature to 104° F. or higher, in children or in old people to 103° F., should in the course of bronchitis, measles, etc., render one very suspicious of broncho-pneumonia.

The *symptoms* of respiratory embarrassment, cyanosis, and a degree of pyrexia out of all proportion to what would be expected in bronchitis without pneumonia, should always, in persons predisposed to it, raise a suspicion of lobular pneumonia.

The *course and termination* are rarely like those of acute croupous pneumonia. Broncho-pneumonia does not, in the majority of cases, terminate from the fifth to the ninth day by crisis, but pursues a protracted, often checkered course of several weeks' duration and ends by lysis. Due attention to the considerations just named, together with physical signs described, ought to enable one to recognize most cases of catarrhal pneumonia.

Differential Diagnosis.—(1) *Acute fibrinous pneumonia* is the affection which, to my mind, is the disease with which broncho-pneumonia is most likely to be confounded. The chief points of difference are: (*a*) Sudden onset with rigor in midst of good health and its greater liability to befall adults. In the old and in children its invasion may, however, be insidious. (*b*) The recognition of signs of uniform consolidation, usually unilateral, and spreading rapidly throughout the lobe. In broncho-pneumonia pseudolobar involvement is the exception and foci are bilateral and multiple. (*c*) Termination is more often by crisis occurring somewhere from the fifth to the ninth day. (*d*) The sputum is rusty and contains pneumococci, while in catarrhal pneumonia of children and aged persons sputum is absent or, if obtained, is scarcely ever other than muco-purulent and may contain influenza bacilli, streptococci or diplococci. (*e*) Herpes is said practically never to occur in catarrhal pneumonia but is common in the croupous form.

(2) *Atelectasis* is a condition that, in children especially, but also in feeble bedridden bronchitics, may be mistaken for lobular pneumonia. It undoubtedly predisposes to and may be called an initial stage of pulmonary inflammation, but the latter is not inevitable, and therefore the following differential points are of importance: (*a*) Collapse is not necessarily accompanied by fever. A low grade of pyrexia may be present, depending on the primary complaint, bronchitis, measles, etc.; but when, in a given case thought to be atelectasis, the temperature is found to be 104° F. or thereabouts, the addition of broncho-pneumonia may be suspected. (*b*) Collapse is changeable under the influence of forced inspiration and change of position. It is well known that atelectatic zones are not constant, but may shift about or be cleared up as soon as bronchial obstruction is removed by cough, whereas, in catarrhal pneumonia, no such mobility of the inflamed foci is possible. (*c*) In atelectasis vocal fremitus and breath-sounds are likely to be absent.

(3) *Small Circumscribed Pleuritic Exudate.*—This condition may give rise to difficulty in differential diagnosis since it may occur in acute infections, e. g., acute articular rheumatism, in the course of which lobular pneumonia may occur. (*a*) Patches of pneumonia are apt to be multiple and bilateral, whereas pleurisy is more likely to be only on one side. (*b*) In pleuritic exudate tactile fremitus is diminished or abolished, while in pneumonia it is apt to be exaggerated, or at least not absent. (*c*) In a pleuritic exudate breath-sounds are diminished or absent and without râles and there may be signs of pressure. (*d*) Exploratory puncture may clear up the diagnosis when all other means fail.

(4) *Acute miliary tuberculosis* is a condition which possesses so many features in common with capillary bronchitis and broncho-pneumonia as to render differential diagnosis most difficult and, in some instances, impossible. (*a*) Both may arise in children, but in young adults acute tuberculosis is relatively more common than catarrhal pneumonia. In such an individual, without preceding acute infectious disease, the sudden development of capillary bronchitis would favor the diagnosis of tuberculosis. (*b*) The detection of crepitus, bronchial breathing and dullness at an apex and the relative predominance of physical signs at the upper rather than the inferior portion of the chest is in favor of tuberculosis. (*c*) If sputum can be obtained its examination may disclose tubercle bacilli or other germs that will determine the nature of the case. (*d*) In acute miliary tuberculosis Ehrlich's diazo-reaction may be present in the urine. (*e*) A splenic tumor would weigh in favor of tuberculosis, except, of course, in cases of typhoid fever complicated with broncho-pneumonia. (*f*) The injection of tuberculine, as suggested by Fraenkel, is applicable only when temperature is normal or not markedly febrile.

Prognosis.—Broncho-pneumonia is at all times a serious affection, and yet, enormous differences are seen in the mortality depending on various factors, as age, cause, constitutional conditions, gravity of certain symptoms, etc.

Age.—Occurring as it does in the majority of cases at the extremes of life when resistance is feeble, this disease is justly regarded as one of the most fatal of pulmonary disorders. Statistics as to the death rate in advanced age are not at hand, but figures from various authors show the mortality in children to vary between 33 and 65.5 per cent. West gives the former figure from 412 cases at St. Bartholomew's Hospital, while Jürgensen gave 58 and von Ziemssen 52 per cent. Of 461 cases collected by Holt below the age of three years the total mortality was 65.5 per cent, as follows:

FORMS OF PNEUMONIA.	Cases.	Deaths.	Percentage mortality.
Primary broncho-pneumonia	194	96	49.4
Following bronchitis of the large tubes	29	19	65.5
Secondary to measles	89	56	62.9
" " pertussis	66	54	81.8
" " scarlet fever	7	7	100.0
" " diphtheria	47	47	100.0
" " ileo-colitis	19	18	94.7
" " epidemic influenza	6	1	16.6
" " variola	2	2	100.0
" " erysipelas	2	2	100.0
Totals	461	302	65.5

Furthermore, the death rate is greatly determined by the age of the child. Thus, of 345 cases below the age of five Holt found the mortality to be 66 per cent in the first year, 55 in the second, 33 in the third, 16 in the fourth, 1 in the fifth. Of 22 cases in the first year von Ziemssen reported 11 deaths or 50 per cent, of 29 cases in the second year 10 deaths or 34 per cent, and in the third year 16 cases and 7 deaths or 44.4 per cent. Bartels' figures tell very much the same story and do not need to be given here. It is thus seen that the prognosis grows more favorable with each year of a child's life.

Cause.—The gravity of the outlook is largely determined by the nature of the ætiological factor. Broncho-pneumonia arising from diphtheria and aspiration of foreign bodies, including ether, is extremely fatal, and recoveries are the exception (diphtheria 47 cases, 47 deaths—Holt). Measles and pertussis are responsible for many victims, but much here depends upon the age of the patient. Thus, figures from Bartels are the following: Of 6 cases of measles in the first year 6 died, of 36 between the first and fifth year 14 died, and of 24 between the fifth and tenth years 9 died. Influenza pneumonia among the old is often very fatal, but here much seems to depend upon the severity of the epidemic. As previously stated, the death rate from this cause ranges from 17 to 40 per cent.

Constitutional Condition.—The previous state of health has great influence upon the prognosis, since robust and muscular children stand a better chance of recovery than do very fat or very thin ones (West). Chil suffering from cachexia of whatever sort, as rickets, scrofula and marasmus, furnish a truly alarming rate of mortality from this form of pneumonia.

Gravity of Certain Symptoms.—Of these a very high and continued fever makes the outlook most grave, as do extreme rapidity of respiration and cyanosis. Extensive bronchitis and broncho-pneumonic areas are serious in proportion to their extent, because they correspondingly diminish respiratory capacity and overstrain the heart. Hence, the higher the respiration-rate and the deeper the cyanosis, the worse may prognosis be said to be. Diarrhœa, vomiting, emaciation, great prostration and pronounced pallor, restlessness to an unusual degree, and apathy may all be regarded as bad prognostic indications and in some cases may portend an inevitably fatal issue.

Treatment. Prophylaxis.—We are powerless to arrest or abort a broncho-pneumonia when once developed and we possess no specific means of cure. Consequently, the most rational thing to do is to prevent its occurrence. That prevention is possible is proven by the experience and utterances of pediatrists that catarrhal pneumonia is rare in their private practice, while it is frequent among the children of the poorer classes and in public institutions.

Of prime importance in the direction of prophylaxis against broncho-pneumonia in infants is *prompt attention to every attack of acute bronchitis* no matter how trivial it appears to be. This should be impressed upon every mother and nurse. The nursery should always be kept well aired and at an equable temperature of about 70° F., and as soon as symptoms of a cold appear the rule just stated should be rigidly insisted on and enforced. The bowels should be cleaned out by calomel or gray powder, mild counter-irritants applied to the chest and other measures instituted including the use of expectorants appropriate to the treatment of bronchitis.

Should a child be suffering with measles, pertussis or one of the other acute infectious complaints of childhood especial care must be exercised against draughts or undue exposure to whatever may be likely to aggravate the congestion and irritation of the mucous membranes already existing. The apartment in which such a little patient is confined should be *often and thoroughly ventilated*, the child being removed to an adjoining room of same temperature during the process of airing. If possible, an open fire should be kept burning in the sick-room. The nursery must not be allowed to cool off too greatly at night, or, if this cannot be prevented, the child must be so clothed and watched as to guard against becoming uncovered and chilled.

Holt is of the opinion that broncho-pneumonia secondary to infectious diseases is more or less communicable and that therefore children with this complication ought to be kept isolated from others having the same primary affection. Another precaution against pneumonia in simple or specific bronchitis is that children below the age of three must not be allowed to retain the same position for too long a time. If infants ill with bronchitis are permitted to lie too long on the back or side they thus favor the production of areas of atelectasis through gravitation of bronchitic products and blocking of bronchioles. For this reason a baby should be turned over from time to time or held in the nurse's lap. If the above-mentioned simple precautions are exercised this dangerous form of pulmonary inflammation will not be likely to ensue.

Finally, for sake of emphasis I wish to repeat what has been said concerning the necessity of a well-aired nursery, reminding the reader that germs abound in badly ventilated apartments which then become strong predisposing factors to broncho-pneumonia.

Therapy.—Since no means are known of arresting or curing the pulmonary inflammation, we are restricted to such measures as will assist nature in her struggle against the disease. These consist, therefore, of means calculated to sustain the patient's strength and modify such symptoms as are in themselves destroying chances of recovery.

Hygienic.—The day is past when pneumonic patients were confined in stuffy, overheated apartments lest the admission of cool fresh air aggravate the inflammatory process. It is now beginning to be generally recognized that plenty of fresh air is essential in the combat against germs and the disorders they induce. Accordingly, the rules laid down for the care of the sick-room during the bronchitis are now equally applicable. If possible the chamber should be a large and sunny one, supplied with some safe means of admitting an abundance of pure air.

The rapidity of breathing indicates the call made by the respiratory centers for oxygen which cannot be better supplied than by frequent renewal of the atmosphere surrounding the sufferer. Moreover, only those persons in immediate attendance upon the patient should be allowed in the apartment. The purpose of this restriction is twofold: first, that the air of the room be not quickly exhausted and contaminated, second, that the patient be not disturbed by the confusion incident to the moving about and going in and out of a number of persons. Sleep is necessary to ill people and hence these patients should not be excited or fussed over more than is unavoidable in the ministration to their actual needs. Body and bedclothing must be changed only as often as is necessary for the purpose of cleanliness since the handling incident to change of garments cannot fail to be more or less fatiguing.

Bathing is one of the measures relied on for relief of certain symptoms, but it must be done properly and under the direction of the physician by some one skilled in its use. If this or any other measure causes a baby to struggle and exhaust its small reserve of strength the bathing must be done as gently as possible. Fortunately, it is usually grateful and its beneficial effects offset any harm that may follow its skillful employment.

Nourishment is indispensable to the maintenance of vital powers and should not be made subordinate to medicinal or other measures. It should be of a kind suitable to the age of the patient. For children nothing, of course, is as good as milk, which, indeed, is the case with anyone having high fever. Nursing infants will sometimes refuse the breast on account of their dyspnœa and when such is the case they may be fed through the nose by means of a catheter or by rectum with properly modified milk. Older children may be fed in the same manner with eggnog, meat juice, broths, soups and the like in addition to milk.

Fluids are to be supplied as freely as the circumstances of the case permit. If the child is struggling for air it may refuse to drink since it is unable to hold its breath long enough to swallow the proffered liquid. Fluids are necessary, however, not only to make good the excessive evaporation of moisture incident to the high body-temperature, but also for the purpose of washing out toxins through the kidneys. Hence, if patients will not drink plenty of water, diluted fruit juices, etc., fluids should be introduced *per rectum*. In all forms of acute disease, therefore, it is well to give a couple of times daily an enema of normal salt solution. If introduced slowly and high up into the colon, it will generally be retained, or at least enough will be absorbed to be of material help to the organism.

The same method of introducing nourishment can be practiced when food is refused because of distress occasioned by suspension of respiration in the act of swallowing. It may also be here stated that medicines may be administered by rectum rather than by mouth when, for any reason, their hypodermic administration is objectionable.

Antipyretics.—When in children, in the acute inflammatory stage, the temperature is high, 104° to 105° F., reduction of the fever is usually thought indicated. To this end the simpler and less depressing the antipyretic employed, the better. Consequently, I do not approve of modern synthetic remedies of the coal-tar series, as antipyrin, antifebrin or even phenacetin, notwithstanding the indorsement of so high an authority as Fraenkel, who gives antipyrin. *Cold* is unquestionably the best refrigerant at our command, since it not only lowers body-heat but strengthens heart-action and calms the nervous system.

Infants may be safely put into a bath of 95° F., which may be cautiously lowered to 90° F. or even 85° F., if the state of the peripheral circulation is carefully watched, meanwhile, and friction of the surface of the body is resorted to from time to time. The duration of the bath should not exceed five to eight minutes depending upon its effect, and its repetition should be determined by the rapidity with which the fever regains its former height.

Older children may be wrapped in a cold pack, directions for which do not need to be given here, or they may be sponged or may have ice applied to the chest. I believe the first of these three measures is the most effective, but a cold pack sometimes occasions shivering and much apparent distress. In such an event sponging or the ice-bag is preferable. In whatever manner the cold is applied, its effect is generally excellent. The restless little sufferer grows quiet, its respirations diminish in frequency and it sinks into a refreshing sleep. I recall a truly remarkable result of this kind in a six months baby, following a bath. The action of the kidneys is generally increased by such an immersion in a tepid bath and the soporose condition so often attending high temperatures in children is lessened.

Conservation of the strength is so important, however, that if the employment of such antipyretic measures causes the infant to struggle and exhaust itself, it is my opinion that less harm may result from the pyrexia than from the treatment. In such cases I would advocate the application of ice or sponging, or diaphoretics may be tried, as sweet spirits of nitre or spirits of mindererus. I have known the temperature to be reduced in a two-year-old infant by enemata of cold water and, to all appearances, with excellent results.

Counter-irritants.—Since time immemorial antiphlogistic remedies have been employed for the lessening of pulmonary inflammations. We no longer believe in the vigorous means once used to this end, since our notions concerning the causation and nature of such inflammations have been changed. Nevertheless, most clinicians still believe in the beneficial influence of mild counter-irritants. Though they cannot arrest the inflammatory process, they may ameliorate some of the symptoms.

Henoch highly recommends the application of dry-cups to the chest, even of young infants, and Fraenkel warmly indorses the recommendation. In this country dry-cups are rarely resorted to, I believe, and most physicians rely on sinapisms and poultices or even camphorated oil. They also envelop the chest in an oiled silk jacket which promotes perspiration while at the same time protecting against chilling of the skin.

In Germany great liking is shown for the enveloping of the chest in a cold compress or a Priessnitz bandage. This consists of a strip of thin muslin, or cheese cloth, folded into two or three thicknesses, and of sufficient length to be wrapped about the chest (see treatment of Acute Bronchitis). After being wrung out in tepid or, in some instances, comparatively cold water, it is placed in position and is then covered over by another cloth of flannel and is changed several times daily. Its effect is to moderate temperature somewhat, quiet respirations and lessen cough. It is certainly an excellent application which if not antiphlogistic or counter-irritant in action, seems to ameliorate some of the pulmonary symptoms in a similar way.

Liebermeister is said to recommend extensive and, as it seems to me, rather heroic counter-irritation of the trunk and the lower extremities by means of a huge mustard plaster inclosing the entire lower half of the body. Notwithstanding the remonstrance it occasions on the part of the child it is claimed that it affords marked relief of the chest symptoms. The fumes arising from the mustard are irritating to the patient's mucous membranes and hence Fraenkel's caution against permitting the broncho-pneumonic child to inhale such fumes is a timely one, equally applicable to the use of sinapisms on the chest.

Inhalations.—Theoretically, the inhalation of antiseptic vapors or sprays should be of the greatest utility in counteracting the baneful action of bacteria in broncho-pneumonia or other forms of bronchial and pulmonary inflammation. If they were effective, they would prove the ideal treatment. I must confess to considerable skepticism concerning the value of this therapeutic measure, certainly in children. Adults may draw the atomized solutions deeply enough into the air-tubes to be of some service, but the most that can be accomplished in the case of young children is to saturate the respired air with vapor in the hope that it may exert some modifying effect on the bronchial secretions.

In the acute stage of catarrhal pneumonia there is, in my opinion, real nger of adding to the existing irritation by the use of creosote-vapors or other antiseptic oils in sufficient strength to be theoretically antagonistic to the germs. Therefore, the best inhalation for young children with this pulmonary complaint is steam, i. e., vaporized water or limewater.

This may be accomplished, as everyone knows, by covering the child's bed with a tent improvised out of a sheet and then by means of a small rubber hose allowing the steam to pass from the kettle directly inside the tent. Or the latter may be large enough to cover over both bed and the apparatus for generating the vapor. In some instances the atmosphere of the entire bedchamber may be impregnated with steam.

The breathing of warm moist air in this manner acts in the same way as does a warm moist climate. It soothes the irritable respiratory passages and, by liquefying bronchial secretions, promotes or facilitates their expectoration. Such a simple measure as the above is, I am convinced, of positive utility and often a more efficient expectorant than some highly vaunted drug. *Per contra* the dry air of a steam-heated apartment is irritating and calculated to aggravate cough and decrease expectoration. If a steam-coil is the only means of heating the sick-room, then a basin of water should be kept on the radiator to lessen the dryness of the atmosphere, or, better still, steam should be actively generated in the apartment by means of a spirit lamp and a kettle of water.

Expectorants have the sanction of long usage and doubtless are of value, else their use would long ago have ceased. In broncho-pneumonia their usefulness is certainly limited to the attendant bronchitis, since they cannot modify the pneumonia directly.

If capillary bronchitis or bronchial obstruction seems to call for the administration of expectorant remedies, then only such should be prescribed as will not upset the stomach or merely dry up secretions instead of loosening them and facilitating their expulsion. For young children therefore nothing is better than syrup of ipecac, or, when a preparation devoid of sugar is preferable, wine of ipecac. A child of three years may safely take 5 drops every two, three or four hours, as is thought best.

Demulcent drinks, as a decoction of Iceland moss or of slippery elm bark, are mildly expectorant and may be of service when the child is not distressed by the act of ingestion. More powerful expectorants, as the various preparations of antimony, and nauseous ones, as chloride of ammonium and squills, seem to me objectionable, especially for children, since they are likely to disturb the stomach and still further increase the repugnance to food on which so much depends. Cases may be seen, of course, in which this objection is subordinate to the necessity for a stimulating expectorant like ammonium chloride, and in such a case the remedy should be ordered, but in as agreeable a vehicle as possible.

For adults requiring a remedy to aid in the expectoration of tenacious mucus I recommend hydrochlorate of apomorphin in $\frac{1}{8}$ grain doses or even twice this amount given in capsule every four hours. It will not nauseate in these doses, taken by mouth, and is an excellent expectorant. I do not approve of the iodide salts, grindelia, lobelia, senega, etc., in these cases of broncho-pneumonia, because I believe them more disagreeable than useful and think all that is necessary can ordinarily be accomplished by steam inhalations, compresses to the chest, mild counter-irritation and mucilaginous drinks. It is very easy to overmedicate these patients and thus do more harm than good.

Emetics.—Now and then cases are seen in which great mechanical interference with respiration is occasioned by profuse bronchial secretions. The child is too weak to expectorate them and is exposed, not only to the peril of suffocation, but to the likelihood of still more extensive atelectasis and inflammation. In such straits it becomes necessary to clear out the tubes if possible and about the only means at command is an emetic. For this purpose syrup of ipecac is perhaps the best for young children, although I know of no valid objection to the hypodermic injection of a dose of apomorphin suitable to the age of the child. The indication is for a prompt and safe emetic that will not produce great depression, and apomorphin is certainly such an agent. It goes without saying that there is no use in trying to vomit any child who is so far gone that reflexes are abolished. In such a case an emetic only aggravates the physical depression. First arouse the patient by stimulation, if possible, and then resort to the emetic. At best it is a forlorn hope in the majority of such cases. It is a good rule to withhold emetics in young children, if possible, especially if prostration is marked.

Sedatives are sometimes required for the mitigation of cough and to allay restlessness or promote sleep. *Morphine and opium preparations, even codeine and heroin, must be ordered with very great caution and discretion in every case of broncho-pneumonia.* The objection to their use lies chiefly in the danger of their too greatly blunting the patient's sensibility to the presence of secretions within the tubes, and hence, by suppressing cough, favor the production of fresh areas of collapse and pneumonia. For small children the bromides are better, and yet even these must be prescribed with caution. Nothing is more likely to quiet the little sufferer than a warm bath.

Tonics.—These are indicated in nearly all cases of catarrhal pneumonia because of its inherent tendency to undermine the strength. Probably no remedy of this class can equal *strychnine*, both as a respiratory and a general tonic. It should not, however, be given in too large dosage—$\frac{1}{40}$ to an adult, and corresponding doses to a child, three or four times daily. Unquestionably the best method of administration to an adult is by hypodermic injection. Quinine in small doses is a general tonic, but is depressing in large ones designed to reduce temperature, which, I agree with Holt in saying, it cannot do in broncho-pneumonia. Arsenious acid is also a respiratory tonic which may be useful in less acute cases, but I see no advantage in iron, and do see disadvantages in cases in which the inflammation is still active.

Caffeine is a capital tonic, both general and cardiac, and one worthy of more extended trial than seems to be usually the case. It should be administered hypodermically to feeble adults, and in form of the benzoate or valerianate, since these are far more soluble than the citrate (see this subject in Treatment of Fibrinous Pneumonia). From 1 to 3 grains may be gi to an adult every four hours according to the emergency of the case.

Digitalis and *strophanthus* may be indicated by cardiac feebleness and rapidity, and when so indicated should be prescribed. I believe a tincture of digitalis from which the free fat has been removed is the best preparation for internal use. When its taste is objectionable, it may be given *per rectum.* For hypodermic administration I recommend digitalone, a preparation made by a well-known Detroit house, since it is comparatively unirritating and efficient. Ten minims may be safely injected into an adult every four hours. In the young whose vascular system is still elastic, digitalis is undoubtedly to be preferred to strophanthus, but in aged patients the latter may give better results.

Oxygen is another therapeutic agent which has a place in broncho-pneumonia, especially when capillary bronchitis is marked or extensive lung areas are involved, as shown by rapidity of breathing, cyanosis and restlessness, or by the stupor of carbon-dioxide poisoning. The gas may be given freely and sometimes continuously by allowing it to flow over the nose and mouth so as to be mixed with air. It certainly seems to minister to the patient's comfort, even if it does not materially influence the course of the disease.

Stimulants.—Many physicians hold the opinion that preparations of alcohol are weakening to the circulation by reason of their vaso-dilator action and that the transient stimulation of the heart observed to follow their administration is secondary to vaso-dilatation. I am not disposed to dispute such a contention, but I am very positive in the opinion that, given judiciously, wine, brandy and whisky do undoubted good in cases of broncho-pneumonia displaying profound prostration. Wine is preferable, I think, to the liquors, and of these Tokay is best when its sweetness is not distasteful. Champagne is also highly serviceable. But whatever form of alcoholic beverage is selected it should be given in small amounts and often, rather than in large doses at longer intervals. They certainly are indicated whenever collapse appears imminent. I do not believe they are to be administered indefinitely, but are to be employed in emergencies, in the same manner that a ship in danger of running on to breakers sometimes casts out an anchor to check its course and permit time for other measures to be tried. Ammonia, as the carbonate, is also an excellent stimulant and highly useful. The dose should be 5 to 10 grains to an adult, and correspondingly smaller ones to a child. It should be given frequently.

In every case of broncho-pneumonia *good nursing* and *unremitting attention* are indispensable. Without an experienced and tactful nurse the physician can do practically nothing with a sick child. The mother may be most desirous of nursing her child and may think herself capable, but, generally speaking, she is not so likely to secure submission to orders as is the proper kind of nurse. Finally, in every severe case the physician should see it often, several times daily, if necessary.

Subacute or chronic cases, i. e., cases of secondary pneumonia that run on into weeks or even months, require management along the lines laid down. But it is not so much the fever and the pulmonary symptoms, strictly speaking, which call for mitigation, as it is the necessity of sustaining the heart and strength. A prime essential is nourishment to which everything else

must be subordinated. General and cardiac tonics are likewise indi
must be selected with discretion so as not to disturb appetite and
It is in this class of cases that good nursing is especially necessary.

Change of climate is very often, nay generally, indispensable to
plete recovery of patients who have survived a severe broncho-p
This is so because of the healing and restorative influence of a mil
and sunny climate in which the convalescent may remain out of
many hours daily. By so doing strength seems to be inhaled w
breath, appetite and nutrition are improved, the blood is regenerate
irritable mucous membranes are soothed and healed.

In all cases, therefore, when at all possible, change of climate
made. Among the poor a long journey may be out of the que
even here it may be possible by a little managing to get a child ren
more healthful surroundings. I recall a case in point where the
chronic broncho-pneumonia following measles, living in a damp d
was sent with her mother to a friend's in a smaller city only forty n
from Chicago. There it was possible to give the child sunshine
air, with the result that a few weeks made remarkable improvemen

CHAPTER XIX

PULMONARY ABSCESS AND GANGRENE

Ætiology.—There are so many points of similarity in these two affections that it is believed conducive to clearness if they are considered together. The conditions predisposing to them are practically identical, and hence the determination of the local peculiarities depends upon the nature of the micro-organism responsible for their production.

Bacteria.—The modern conception of disease consequent upon bacteriological investigations has cleared up much that was before obscure concerning the causation of abscess and gangrene of the lung. There are still some questions remaining unanswered, however, which will be touched upon incidentally in the following paragraphs.

The presence of micro-organisms is indispensable to the production of suppuration within a circumscribed area in the lungs. In some instances, perhaps, an intense nutritive disturbance prepares the soil for the action of the germs; in others the germs themselves exert so malign an action that this leads to death of the part. The same remarks hold true of gangrene, but it is likely that a serious interference with local nutrition precedes in the majority of instances.

As might be expected, the organisms responsible for abscess are most commonly pus-cocci, and yet, it is likely that pathogenic bacteria may, when unusually virulent, cause necrosis and suppuration. Of these latter, the pneumococcus and the influenza bacillus are the most frequent. In numerous instances which were studied with special reference to the germs of the abscess cavity or sputum these were found to consist of mixed forms.

In gangrene also the bacteria are found to be numerous and diverse. Fraenkel is of the opinion that the necrosis and suppuration are due to pyogenic organisms, while the putrefaction is owing to the presence of other bacteria which either have not been definitely identified or are of several kinds. These saprophytes are long slender rods which, because of their filamentous processes, closely resemble leptothrix. Leyden and Jaffe discovered a form which, because of its resemblance to the leptothrix buccalis, was termed by them leptothrix pulmonalis. Stained with a diluted tincture of iodine this organism assumes a blue or violet color. In some instances bacteria were also discovered by these investigators which showed a mushroom-like or spiral formation.

Reinbach, in a case of diffuse gangrene of both lower lobes in a child, discovered a bacillus which resembled that of anthrax but which, according to Fraenkel, possessed only a morphological similarity, since its cultural peculiarities were not demonstrated. Hirschler and Terray, in three cases of gangrene, in addition to various staphylococci, micrococcus tetragenus, and bacillus pyocyaneus, discovered a peculiar kind of coccus which grew in ordinary culture-media and gave forth an odor of putrescence very like that of gangrene.

Babes is said (Aufrecht) to have found two kinds of bacteria one of which resembled the bacillus œdematis malignæ, while the other was very like the bacterium coli commune, excepting that it liquefied gelatine. Experimentally it was demonstrated that these organisms were capable of producing gangrene, but that in cultures they rapidly lost their virulence.

Guillemont believes that gangrene of the lungs may be caused by various anaérobes and among such he attaches especial power to the bacillus racemosus. Aufrecht agrees with Fraenkel in the conclusion that the specific organism of pulmonary gangrene has not yet been determined and that these bacteria, of whatever kind they may be, can become operative only in the presence of other micro-organisms already existing in the lung. The conflicting results of bacteriological investigations may well lead one to query if putrefaction of the necrotic area may not result from the action of several different sorts of germs.

Predisposing Causes. *Acute Fibrinous Pneumonia.*—This affection is complicated by abscess and gangrene of the lung less frequently than is generally supposed. Such was the belief of Laennec and his immediate followers, and statistics bear out their assertions. Thus, of 424 autopsies on pneumonia patients by Aufrecht and Kerr, abscess of the lung occurred 8 times, a percentage of 1.9, while of 750 cases of pneumonia Sello discovered 11 instances of abscess or 1.5 per cent. As regards the infrequency of gangrene, Fraenkel states that he found it in but 0.4 per cent of his cases of pneumonia. Kerr found but 2 instances of gangrene in 171 cases of pneumonia, which is 1.17 per cent. In 5,072 necropsies Hensel met with pulmonary gangrene but 83 times, and of these pneumonia was the cause in only 14 instances. On the whole, therefore, both complications are rare and occur in about the same relative frequency.

Such is the rarity of abscess of the lung in the course of acute fibrinous pneumonia that some authors are inclined to doubt its occurrence in this form of the disease. Thus, Sonnenberg, judging from his own experience, inclined to the belief that when abscess develops it is in influenza and not pneumococcus pneumonia. Such a view is too radical, however, since there are many trustworthy observations by competent clinicians which prove conclusively a direct, albeit rare, ætiological connection between acute fibrinous pneumonia and the form of lung destruction now considered.

Of greater importance is the query concerning the conditions which cause a given case of pneumonia to terminate in suppuration or gangrene. Probably the intensity of the local inflammation is of great moment, but inasmuch as these complications are observed more often in the so-called asthenic than in the sthenic type of pneumonia, it is the degree of tissue resistance or the admixture of other germs more than the primary infection that is responsible for the mishap. Certain it is that abscess and gangrene are seen more frequently in the alcoholic and enfeebled than in the robust. As previously stated, the pneumococcus alone is capable of exciting a destructive inflammation, and if we assume a low state of local as well as general nutrition it becomes easy to understand the relatively frequent development of abscess or gangrene in drunkards and other debilitated subjects.

It is stated by some writers (Wilson Fox, e. g.) that abscess is particularly apt to befall the upper lobe, a circumstance attributed to the likelihood of a previous tuberculous invasion of this portion of the lung and consequent malnutrition of the part. When we consider the relative immunity of the upper lobes to pneumonia as compared with the lower, it appears peculiar indeed for abscess to find its favorite seat at the apex. On this account I am inclined, while not denying the correctness of Fox's statement, to doubt if the cases were in reality instances of acute fibrinous pneumonia as often as was believed. Acute tuberculous broncho-pneumonia of the apex is so common and is so difficult of clinical differentiation from croupous pneumonia as to make it not unreasonable to infer that when abscess in this situation was observed by earlier clinicians it was many times of tuberculous and not of pneumococcus origin. Finally, as respects the occurrence of gangrene in the asthenic type of pneumonia, Lenhartz makes the very pertinent suggestion that it is in reality an aspiration pneumonia, or, in other words, an infection of the already inflamed lung in consequence of the aspiration of secretions of food particles from the mouth and throat which carry with them putrefactive organisms. Whether or not such is in reality the case, it is not at all impossible and renders very pertinent the suggestion to take particular pains in the hygiene of the mouth in every case of acute pneumonia, especially when the patient is delirious or greatly debilitated.

Acute Broncho-pneumonia.—This form of pulmonary inflammation may also terminate in abscess or gangrene, especially when of influenzal origin (Sonnenberg, Fraenkel). Yet, as will be seen later on, most instances of lobular pneumonia thus terminating are, in reality, cases of so-called aspiration or deglutition pneumonia. In such, the pulmonary organs suffer traumatic damage while at the same time putrefactive and pyogenic organisms are carried along with the inhaled substance. In other cases the circumscribed inflammation is a sequence of pulmonary embolism which, as will now be seen, is a powerful factor in abscess and gangrene.

Infective Emboli.—These are relatively frequent and well-recognized causes of either abscess or gangrene according to the characters of the bacteria concerned. Thus, of 83 cases of pulmonary gangrene analyzed by Heschl, embolism and thrombosis were put down as the cause in 20, while of 29 instances in which the causation was determinable, Coupland found 4 that could be referred to the same condition. The diseases responsible for pulmonary infarcts are numerous, but puerperal sepsis is one of the most common. In septic metritis or endometritis, as well as in other suppurative processes, venous thrombosis is set up and, the coagulum not being firm, fragments are broken off which are carried either to the lungs directly or to other organs. In the latter event abscesses are formed which may, in turn, give rise to septic infarcts in the lungs (Fig. 35). There may be but a single focus of suppuration in the pulmonary parenchyma, but often there are multiple abscesses of variable size.

I recall the instance of a young woman at Cook County Hospital who died from multiple pulmonary abscesses secondary to metritis resulting from abortion. The initial symptom was a sharp pleuritic pain just above the liver, on the right side, followed by cough and rise of temperature. Positive findings on the part of the lungs were never discovered, and yet the necropsy disclosed numerous infarcts with surrounding small abscesses.

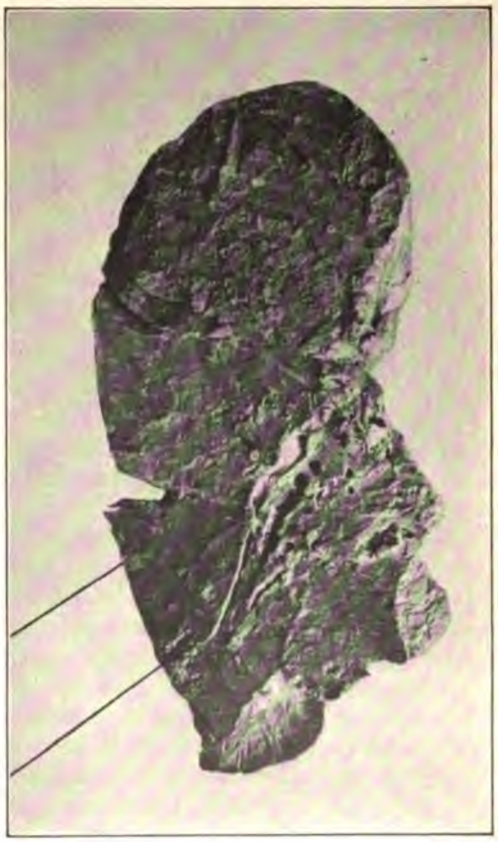


FIG. 35.—Small embolic abscesses in lung.

Other diseases sometimes responsible for pulmonary infarcts of a septic nature are ulcerative endocarditis, chronic otitis media, erysipelas and suppurative inflammations within the intestinal tract, as rectal abscess or ulcer. In the case of septic endocarditis it is not necessary for the process to be located in the right heart, for emboli may be given off from the mitral or aortic valves and, being carried in the blood-stream, may pass through the arterioles and capillaries to reach, at length, the capillaries of the lungs.

In the case of middle-ear disease, thrombosis may occur in the sinuses of the petrous portion of the temporal bone or in the internal jugular vein, whence infective particles finally reach the lungs. Leyden is of the opinion that when pulmonary abscess or gangrene results from chronic otitis the infective agent reaches the lung by means of the blood-stream, even when thrombosis cannot be demonstrated, since this is far more likely than is the discharge of pus from the Eustachian tube, as has been suggested.

In a male patient who died at Cook County Hospital with clinical and *post-mortem* evidences of multiple areas of gangrene scattered throughout the right lung, there was found a septic thrombosis of the right internal jugular vein secondary to an old middle-ear disease. The clinical findings were those of multiple foci of broncho-pneumonia without demonstrable cavity formation but with the characteristic fœtor of sputa and breath.

If rectal abscess or pylephlebitis be the ultimate cause of the pulmonary infarcts, infective emboli may pass directly from the veins of the portal system to the capillaries of the lungs, or they may first give rise to multiple foci of suppuration in the liver and these in turn may occasion embolic necrosis and suppuration within the pulmonary parenchyma (Fraenkel).

Foreign Bodies.—Under this head must be considered those instances of aspiration or deglutition pneumonia which result in suppuration or putrefaction according to the kind of organisms introduced with the substances. A great variety of conditions may be responsible for these pneumonias. Thus pus or fragments of tissue may be aspirated into the lungs during operations on the throat or larynx; or innumerable bodies of various sorts may be inhaled (see Foreign Bodies in the Bronchi), as particles of food breathed in by the apoplectic, the insane or feeble bedridden patients, etc. In all such cases, pyogenic or putrefactive bacteria gain access to the lungs along with the foreign materials; or the retained bronchial secretions, themselves infected, excite an intense inflammation which results in necrosis and suppuration. Foci of this kind may be single and of considerable dimensions, but are sometimes multiple.

Abscesses of Adjacent Structures.—Among the rare causes of pulmonary abscess must be mentioned suppuration of contiguous organs, e. g., hepatic and subphrenic abscesses which break through into the bronchi. Empyema and suppurating bronchial or mediastinal glands must also be included in this list. Unquestionably these and other collections of pus may rupture into the bronchi and be discharged without causing pulmonary abscess or gangrene; but now and then the infective material is aspirated into neighboring lobules with disastrous consequences. They come properly, therefore, under the head of cases of aspiration pneumonia.

Tumors.—These may be situated within the thorax when they probably exert their effect through pressure, or they may have their seat in the tongue, as seen in ten of Heschl's and four of Coupland's cases. In cancer of the tongue the gangrene results from inhalation of oral secretions or other substances. Among the intrathoracic tumors may be enumerated aortic aneurysm, cancer of the œsophagus, mediastinum or lung. The exact mode of production of pulmonary gangrene in these cases is still not quite clear. By some writers it is attributed to a pressure pneumonia or to occlusion and thrombosis of a pulmonary artery, this latter being by Fox thought not unlikely in instances of aneurysm.

Fowler is of the opinion that the gangrene may be due to retention and decomposition of bronchial secretions, particularly if bronchiectasis has resulted from pressure by the tumor. In support of his contention he cites the instance of a mediastinal lympho-sarcoma which terminated in gangrene and in which the left lung was found in a state of sacculated bronchiectases with septic pneumonia.

Bronchiectasis.—That this disease not infrequently leads to gangrene of the lung is shown by five instances in Heschl's table and two in Coupland's.

The explanation is to be found in the retention of the bronchial secretions and their contamination by germs of decomposition. But how these latter gain access to the bronchiectatic cavities is not quite clear unless by aspiration from the mouth in the act of coughing, which may be so violent and prolonged as to necessitate vigorous inspiration at its close if not during its continuance.

Pulmonary Tuberculosis.—One frequently hears of cases of acute phthisis which display the clinical features of an abscess of the lung, i. e., there is a sudden expectoration of a large quantity of purulent material with subsequent signs of excavation. Strictly speaking, these are instances of acute caseation, but whether induced by the tubercle bacillus alone or by pyogenic organisms either alone or in conjunction with the bacillus is not always quite certain.

The occurrence of gangrene in the course of pulmonary tuberculosis is very exceptional. As with medical statistics in general the figures on this head are somewhat conflicting. Thus, Boudet is said to have found gangrene but once out of 160 cases of phthisis, whereas Fox discovered 6 instances among 99 cases of pulmonary tuberculosis that came to necropsy. It is interesting to note, however, that of 10 instances of gangrene of the lung, tuberculosis in other parts of the organ existed in 5.

The cause of the putrefaction in such cases is obscure. The complication occurs most often in cases of vomica or bronchiectasis and hence is probably owing to retention of the contents of the cavity and its infection by saprophytic bacteria derived in some manner from the oral mucosa. An odor of decomposition may sometimes be perceived in the breath of tuberculous individuals, but the conclusion is not necessary that gangrene is present (Fowler). For some reason gangrene is most likely to occur in cases of fibroid phthisis, possibly because of the liability to bronchiectasis in this class of cases.

Trauma.—It is not singular that perforating wounds of the chest should be followed by abscess or gangrene of the lung, for in some of such instances foreign bodies are carried into the lung along with the bullet and are there left. Thus, I know of a case of a soldier who coughed out a piece of his coat along with a quantity of pus several months after his wound was received. But under the present heading are grouped those cases which have displayed abscess or gangrene of the lung as a result of injury to the chest-wall without laceration. In some a pneumonia first became apparent and this speedily underwent suppuration or gangrene.

In other cases the lung mischief has not made its appearance until a number of weeks subsequent to the accident. As no other ætiological factor could be elicited it is deemed but fair to ascribe these cases to the contusion of the thorax. Why trauma of this kind should lead to abscess or gangrene is still a mystery unless, as assumed by Fraenkel, a solution of continuity within the pulmonary parenchyma does actually occur which favors the action bacteria that are present or subsequently penetrate to the area whose resist- ce has been undermined.

Morbid Anatomy.—The appearances vary considerably in the two proc- and in accordance with the causation. The abscess area presents a sh-gray color, while that of the gangrenous zone is dirty gray when recent and later on brownish or greenish-black from admixture with blood. Either process may be single or multiple (Fig. 35) and of variable size. If it occurs in the course of fibrinous pneumonia it is more likely to be single and of considerable extent, in some cases even as large as a child's head. Under such conditions an abscess may at first sight be taken for an area of gray hepatization but on closer examination is found soft and easily broken down. It is apt to stand out more sharply from the surrounding zone of red hepatization. In outline the necrotic area whether suppurative or gangrenous may be either regular or irregular.

In cases due to embolism there may likewise be but a single focus of necrosis but multiple areas are not uncommon. They are wedge shaped and vary much in size, depending upon the caliber of the plugged vessel. If the process be merely suppurative it is soft and creamy, wanting in the normal structure of the pulmonary parenchyma in consequence of infiltration with an immense number of leucocytes that have undergone coagulation necrosis. In gangrene the area, also soft and dry at first, soon becomes pulpy and structureless and in consequence of the added putrefaction emits an intensely disagreeable fœtid odor like that of decomposing flesh and fæces combined.

In both processes alike, the surrounding lung tissue is hyperæmic and œdematous, while the bronchi leading from the necrotic focus display evidences of more or less intense inflammation. If the diseased area is situated near the periphery of the lung the overlying pleura is seen to be inflamed and covered with exudate.

Examined with aid of a microscope the diseased focus is seen to be made up of disintegrated pulmonary tissue with a multitude of leucocytes in the process of disintegration and of micro-organisms. In abscess the observer may recognize elastic fibers or even considerable masses or shreds of lung substance, whereas in gangrene these may not be distinguishable by reason of their having been destroyed through the peptogenic action of the ferment generated in the process of putrefaction.

In very many instances the necrotic and suppurative or putrefactive material has been more or less completely evacuated through the bronchi leaving a cavity behind of variable size and conformation. The walls of this cavity are composed of the inflamed and œdematous or hepatized lung, as the case may be, and their surface is ragged from masses or shreds of necrosed or putrefied tissue in which may be detected stumps of eroded bronchi and blood-vessels. The vomica is rarely entirely empty but contains a variable amount of purulent or putrid material, some of which latter is also apt to be found in the congested or inflamed air-tubes.

In more chronic cases the cavity becomes lined with a false membrane of granulation tissue or may even be surrounded by a newly formed connective tissue. In cases of chronic abscess this lining membrane continues to secrete pus, while in gangrene it is apt itself to fall victim to necrosis and permit gradual extension of the gangrenous process. It is because of this tendency to steady advance of the necrotic process that gangrenous cavities rarely heal spontaneously. In abscess, on the contrary, it is quite possible for the suppurative process to cease in time and for the cavity to become obliterated, if not too large, by cicatrization and contraction, or even to undergo calcification. The surrounding lung is likely to be in a state of compensatory emphysema.

In exceptional instances a lobe or an entire lung may be found in a state of diffuse gangrene. It is soft and pultaceous, of a dirty gray or greenish-black color and may or may not emit a fœtid odor. The necrotized tissue is not walled off by hepatized lung but merges gradually into inflamed or œdematous parenchyma. There may or may not be cavities and the visceral pleura shows the changes of inflammation. This diffuse form is thought to result from aspiration of septic substances into various parts of the lung.

Other *post-mortem* findings consist in the changes of the primary affection and in various complications that have resulted from emboli given off from thrombosed pulmonary veins. Among these latter are abscesses of the brain, liver, spleen, kidneys or other organs. Cerebral abscess is the most important of these complications from a clinical standpoint since it leads to paralysis. It is most commonly single but Netter has directed attention to the comparative frequency with which brain abscess in the course of pulmonary gangrene is multiple. The most usual seat of septic emboli is in the artery of the fossa sylvii.

Symptoms.—Excepting as regards the sputum and the more intense prostration attending gangrene the two affections now considered bear a close resemblance in their general clinical features. They may owe their origin to similar causes and yet present some clinical differences. The onset in both may be disguised by the symptoms of the primary disorder, as in acute pneumonia, or the involvement of the lungs may be declared by symptoms referable to these organs primarily, after which signs of broncho-pneumonia may or may not be discovered according to the seat and extent of the process.

If it be an acute pneumonia which is responsible for the abscess or gangrene it need not necessarily display any features at first that arouse a suspicion of the destructive process going on in the affected lobe. As the pneumonia proceeds, however, crisis does not take place and the patient shows great prostration. Dullness persists, the breath-sounds remain bronchial or at length become feeble or inaudible over a limited area and râles of resolution fail to appear.

Cough remains frequent or perhaps grows more severe. If the patient is very asthenic and delirious, as is not infrequent in drunkards, it may remain an inconspicuous feature. At length, in most instances about the end of the second week, there is a more than usually violent paroxysm of coughing and a quantity of pus is suddenly expectorated which, on minute inspection, is found to contain elastic fibers and shreds of pulmonary tissue. Or the breath of the patient begins to take on a fœtid odor and the sputa assume the brownish, greenish or blackish color and other features of gangrene. In some cases of abscess the patient's condition now begins slowly to mend and at length, after a tedious convalescence, he regains health.

In other cases, especially in gangrene, the state of the individual does not improve, but on the contrary grows steadily worse. Chills, irregular fever and sweats occur, emaciation and prostration increase and the cond becomes plainly that of profound sepsis. If the process be single and extensive signs of cavity are detected, or, as in a male patient in Cook County Hospital in whom the gangrene was the result of thrombosis of the internal jugular from chronic otitis media, there may be multiple foci of bronchopneumonia but without demonstrable cavities.

If in these cases, whether of suppuration or putrefaction, the area is not accessible to surgical intervention or the process does not become arrested by elimination of the necrosed tissue through the bronchi, the condition either terminates speedily in death or passes over into a chronic one of abscess or gangrene cavity of greater or shorter duration. In some cases the contents of the cavity are expectorated so easily and freely that absorption of toxines is limited and symptoms of sepsis are moderate. In other instances the state of the patient is markedly septic and leads inevitably to death. Such is particularly the result in gangrene. In still other cases, especially of abscess, toxic symptoms are subordinate to those of cough, anorexia, debility, etc., but unless nature or the surgeon brings about a cure the strength of the patient gives out and death ultimately triumphs.

A typical instance of this kind was in a young lady of twenty whom I saw, in consultation with Dr. C. E. Bragdon of Evanston, Illinois. She had been taken several weeks earlier with a primary croupous pneumonia of the right lower lobe which ran a severe and protracted course. At length, without any special symptoms to point to the impending rupture of an abscess a large amount of pus was expectorated directly following a violent fit of coughing. For a time thereafter the general symptoms improved and the lung began to clear up. Cough persisted, however, and became intense and of a paroxysmal character, which prevented adequate sleep and necessitated utmost quiet of body lest cough should be excited. The expectoration remained purulent, although in lessened amounts, strength failed alarmingly and emaciation became pronounced.

The pulse grew more rapid and feeble, nervousness became a marked feature, and to her friends the young lady seemed "passing into a decline." Signs of cavity were not positive and yet, from the pectoriloquy and tubular breathing over a circumscribed area in the middle third of the right lung, I was able to locate a small vomica with tolerable certainty. By means of forced feeding and other therapeutic measures the patient's condition was made to improve slowly, and at length, after a convalescence of many weeks, to terminate in complete resumption of health. Moderate dullness persisted and the right side became slightly retracted, but a year subsequently the young lady was to all intents and purposes entirely well.

When the abscess or gangrene is of embolic origin the clinical features depend upon the number and extent of the necrotic foci. If they are multiple and small, as is apt to be the case in puerperal sepsis, their presence may be concealed by the symptoms of the primary affection. This is certainly true of abscess, whereas gangrenous areas are likely to reveal their existence by the characteristic fœtor of the breath if not by typical sputum. In cases of numerous small areas of suppuration superficially located the only symptoms indicative of lung involvement may be sharp pleuritic pain, cough, acceleration of breathing and increase of fever. Such was the paucity of symptoms in the case of puerperal metritis seen in Cook County Hospital, and already mentioned under ætiology.

In fortunate cases the pulmonary embolism may lead to a considerable patch of pneumonia which at first may be mistaken for a primary inflammation of pneumococcus origin. Fraenkel narrates the instance of a young woman who developed a pneumonia of embolic causation in the left lower lobe. Crisis did not occur, although on the ninth day it seemed imminent, but instead, on the thirteenth day, she suddenly expectorated a large amount of pus which was succeeded by definite signs of cavity in the left lower lobe behind. Improvement in symptoms followed the emptying of the cavity and recovery ultimately took place.

Analysis of Symptoms. *Fever.*—In cases of abscess or gangrene that arise in the course of some affection already producing pyrexia the temperature curve may remain not appreciably affected, or it may manifest modifications plainly referable to the lung mischief. In other instances the febrile reaction is due to the initial pneumonia, e. g., aspiration pneumonia. As suppuration or putrefaction takes place the fever is likely to assume a higher general average or to display the irregularity indicative of sepsis. This is especially true of gangrene.

After evacuation of the pus by whatever means the temperature is likely to decline and may, when suppuration ceases or the discharge of the purulent material is free, gradually return to normal. Not so, however, with gangrene. Here the absorption of septic products persists, although to what extent depends upon the discharge or retention of the putrid material. In diffuse or multiple gangrene in which excavation does not occur symptoms of blood poisoning are profound and pyrexia of a hectic type persists to the end.

Chills and Sweatings.—These, as would be expected, accompany the process but vary in degree. They may be marked in some cases of suppuration, even as in gangrene, but for the most part are scarcely likely to be so intense. In cases in which the necrosis of the lung is attended with putrefaction all the phenomena of sepsis are likely to be intense and hence may be regarded as a measure of the malignity of the infection. Accordingly, one case may show but slight shiverings and moderate perspirations, while another may display intense rigors and drenching sweats which, like the fever, may recur one or more times daily.

Cough.—This symptom is rarely if ever absent but varies greatly in frequency and violence. At first, in pneumonic cases, it is attributable to the primary process and hence, in the asthenic pneumonia of drunkards, may be insignificant. In embolic processes also cough is present, but aside from its frequency may not be at all peculiar. When, on the contrary, this feature of a case is more than ordinarily distressing and violent it is due probably to associated bronchitis. The ichorous material which passes over the bronchial mucosa sets up inflammation and this very naturally excites cough. Other things being equal, the more difficult the expulsion of the offending material, the more violent and spasmodic is the cough. Finally it is likely that the sensibility of the individual exerts much influence over the prominence of this feature of the case.

Expectoration.—This is the most striking and characteristic symptom of both abscess and gangrene. In the former affection it consists of a more or less creamy material which, examined microscopically, is made up of pus cells, lung tissue, cellular débris, bacteria and hæmatoidin crystals. Its color is variable. In acute pneumonia it may be of a distinct green hue for a few days preceding the discharge of pus, and, when it occurs, should arouse a suspicion of impending suppuration, although not invariably so (Traube). Lenhartz and Fraenkel both mention a brownish tint of the sputum which gives it a resemblance to the crust of Semmel or German breakfast rolls, while Trousseau described pus from pulmonary abscess that was distinctly chocolate colored. Exceptionally, also, it may have a bright yellow or ochre hue from its being stained with biliary coloring matters.

But whatever its appearance, its special characters which distinguish it from the expectoration of other pulmonary affections are its sudden evacuation during a paroxysm of cough and its containing the ingredients mentioned above. In particular it is the presence of hæmotoidin crystals, elastic fibers or fragments of pulmonary parenchyma which render certain its origin in necrosis and suppuration. It is most important, therefore, that the sputum first discharged be minutely examined with the aid of a microscope, for after the evacuation of the necrotic area the sputa may no longer contain lung tissue but only pus, crystals and bacteria.

In pulmonary gangrene the expectoration is both like and unlike that of abscess. It is similar in the beginning on account of its containing elastic fibers or fragments of lung tissue, the elastic fibers either being clearly recognizable as such or but indistinctly showing a network arrangement, i. e., the peculiar alveolar structure. In more chronic cases pulmonary tissue may be wanting because, as first pointed out by Filehne, of the peptogenic action of a trypsine-like ferment which dissolves the elastic fibers. In such cases, therefore, the resemblance to abscess sputum is lost and its dissimilarity seems to stand forth even more conspicuously.

The special characters which distinguish the expectoration of gangrene from that of abscess are its *fœtor* and its *color.* The sputum of suppuration is usually odorless, whereas that of putrefaction emits a peculiarly offensive odor which scents the patient's breath and makes the examination of his chest a very trying ordeal. In the sputum-cup the expectoration is often well-nigh intolerable. This fœtor is due to the presence of skatol, which is a product of decomposition of animal proteids and hence gives to the sputa a smell like putrid flesh and fæces combined. So offensive is this fœtor that it may sicken the patient himself and permeate the atmosphere of the entire apartment.

The color of gangrenous sputum is also distinctive from that of suppuration and all other pulmonary affections, save fœtid bronchitis. When allowed to settle in a sputum-glass it divides into three layers, the uppermost of which is made up of froth having a dirty grayish color. The second or middle stratum consists of brownish or yellowish-brown serum holding flocculi of mucus in suspension, while the bottom layer of a brownish-black or greenish-black appearance is thick, and composed of detritus of pulmonary parenchyma, bacteria and numerous other substances. Among these various ingredients are the hæmatoidin crystals previously mentioned which appear as beautiful golden-brown plates and starlike forms, also oil-drops and crystals of the fatty acids. In some cases also are discoverable the so-called *Dittrich's Plugs* which are minute or bean-sized bodies of a yellow-brown color. Examined microscopically they are seen to be composed of soft granular débris, oil-drops, crystals and bacteria. These plugs emit a horribly stinking odor and are responsible for the fœtor of the sputum.

Hæmoptysis.—This is not usual in either suppuration or putrefaction of the lung, but inasmuch as the necrotic process may lay bare vessels of considerable size, a pulmonary hæmorrhage is not impossible, especially in gangrene. This liability should always be kept in mind, therefore, whenever the necrotic process is extensive or advancing.

Pain.—This may or may not be a symptom of either of the affections now under consideration. It is complained of whenever the diseased area is so superficial as to involve the pleura and then displays the characters of ordinary pleuritis. Exceptionally, pain is a prominent feature and, according to Lenhartz, enables the patient to indicate the seat of the necrosis.

Prostration and Emaciation.—These are always present in greater or less degree, depending upon the extent and special characters of the lesion. Hence they may be said to stand in direct relationship to the intensity of the septicæmia, that is, the pyrexia, sweating, anorexia, diarrhœa, etc., which accompany every case of sepsis.

The *urine* is likely to display the changes usually found in febrile diseases, and hence the more profound the infection, the more marked are the albuminuria, casts and other alterations seen in acute nephritis of this causation.

The *pulse* answers in frequency and strength to the severity of the local disease and its inroads on the vitality of the individual. Such other features as the case may exhibit are either such as might naturally be expected from the progress of the affection or such as are produced by complications, in particular, by cerebral abscess. Accordingly no attempt is made to describe them here.

Physical Signs.—There is nothing in examination of the chest to indicate the destruction going on within the lungs. The findings are either such as belong to acute fibrinous or acute broncho-pneumonia or, after evacuation has occurred, are such as belong to cavity from whatever cause. In the case of pulmonary infarcts there may be no discoverable physical signs at all.

Inspection.—This is of little if any value, save in so far as the accelerated, difficult or superficial breathing may assist in the detection of lung involvement in obscure cases, e. g., pulmonary embolism in the course of a phlebitis, puerperal sepsis, etc.

Palpation.—This may be normal or modified according to circumstances. Small or deeply situated foci of necrosis may produce no appreciable alteration of pectoral fremitus, whereas, over a pneumonic area of considerable extent fremitus is likely to be increased. When, on the other hand, the inflamed area has actually suffered necrosis vocal vibration may be diminished or absent. Palpation is of aid in most instances, therefore, in locating the seat of the lesion. This is particularly true when cavity has formed, since circumscribed exaggeration of fremitus is then likely to be discovered.

Percussion.—This may or may not detect dullness over the site of the process. Accordingly, the extent and degree of dullness depend upon the dimension and location of the focus. After evacuation of the broken-down material, percussion is likely to reveal the changes in the pulmonary note indicative of cavity. For particulars the reader is referred to the Diagnosis of Pulmonary Cavity in the chapter on Pulmonary Tuberculosis.

Auscultation.—Such changes in the breath and voice sounds as may be detected are dependent upon the size, situation and number of the diseased foci. Pneumonic areas give rise to bronchial breathing the intensity and distinctness of which bear direct ratio to the extent and superficiality of the pneumonic patch. In acute lobar pneumonia the breath sounds over the necrotic area may remain tubular or become enfeebled, while the voice sounds are correspondingly altered. There may be no auscultatory evidence of the suppurative or necrotic process, but should a circumscribed area fail to transmit the sounds elicited elsewhere this may be in consequence of its having undergone destruction and lost its power of conducting sounds. Ordinarily, however, auscultation furnishes but scanty information before the stage of excavation. Then the breath sounds and voice display the modifications found in vomica, amphoric breathing, cavernous voice, etc., for a full description of which the reader is referred to Pulmonary Tuberculosis and that portion of the subject dealing with Diagnosis of Cavity. Abscess or gangrenous foci of embolic origin, especially when scattered and small, are likely to occasion no recognizable changes in the auscultatory findings.

Diagnosis.—From what has just been said concerning physical signs it is evident that the existence of either abscess or gangrene of the lung must be diagnosed from other data than those furnished by a chest examination. In many cases of suppuration its recognition is wholly impossible or purely inferential before the appearance of the characteristic expectoration. In gangrene a diagnosis may sometimes be made from fœtor of the breath, even before characteristic sputa appear. Caution is essential in such a case, however, since the condition may be a fœtid bronchitis or there may be merely decomposing blood in the air-passages. On the whole, therefore, diagnosis cannot be positive without the data furnished by the expectoration. This statement holds true especially of abscess.

In suspected cases particular attention should be paid to the anamnesis. A history of injury to the chest wall and still more of aspiration of a foreign body strengthens the assumption that the pulmonary symptoms are due to destructive inflammation. Likewise, symptoms on the part of the lungs arising unexpectedly in the course of a suppurative process in other parts of the body, in a paralytic or in the insane, after an operation on the throat or tongue or in conjunction with cancer of these parts, chronic otitis media, etc., should always lead the physician to entertain the likelihood of either abscess or gangrene.

Should the examiner in any such case detect signs of localized pneumonia, he must regard the process with apprehension until its outcome in recovery or the evacuation *per os* of characteristic sputum determines its exact nature. In every instance, however, the diagnosis must remain a matter of more or less uncertainty until the appearance of sputa containing the ingredients previously described, in particular, crystals of hæmatoidin.

The recognition of abscess or gangrenous cavities must depend on the detection of physical signs of vomica together with the history and symptoms, especially expectoration. In doubtful cases the X-ray may yield valuable information, and should be tried.

Differential Diagnosis.—This concerns first, the differentiation of abscess from gangrene, a matter of importance in some cases as regards prognosis. Doubtless an abscess cavity may become infected with germs of decomposition and be converted into one of gangrene, but, as a rule, there is no difficulty in distinguishing the two affections if the special characters of the expectoration be observed.

Abscess of the lung must be differentiated chiefly from the three following diseases, bronchiectasis, empyema with rupture into a bronchus, and acute pulmonary tuberculosis.

(1) Bronchiectasis. This is differentiated by (*a*) its chronicity and gradual development after some acute or chronic lung disease, as bronchitis. (*b*) Septic phenomena are usually insignificant or absent and the general health is less affected. (*c*) The expectoration is different both in its mode of evacuation and in its characters, particularly the absence of hæmatoidin crystals and fragments of lung tissue, unless, in exceptional cases, ulceration of the lung results when the condition has become converted into one of abscess. (*d*) Signs of cavity are more likely to be obscure or are dispersed throughout various portions of the cirrhotic lung.

(2) Empyema with rupture of pus. Under some circumstances this may be distinguished from abscess only with great difficulty or not at all. The points of difference are the following: (*a*) Empyema is more frequent in the course of or after a croupous pneumonia, and is not a sequel of the other conditions that predispose to abscess. (*b*) The rupture into the bronchi is generally accompanied by sharp pain in the side and the pus does not contain the ingredients seen in necrosis and suppuration. (*c*) The local signs following evacuation of the pus belong to those of pneumothorax rather than to those of vomica. (*d*) Before rupture occurs the degree of constitutional disturbance is rarely so pronounced as in abscess.

(3) Acute pulmonary tuberculosis. This affection is likely to resemble only that form of suppuration occurring in acute fibrinous pneumonia. The points of difference are, (*a*) the process is almost always apical. (*b*) If a focus of caseation is suddenly expectorated, forming a tuberculous abscess, the pus contains tubercle bacilli along with débris of lung tissue. (*c*) Tubercle bacilli usually continue to be expectorated after the disappearance of disintegrated lung substance.

Pulmonary gangrene must be differentiated from two affections, fœtid bronchitis and bronchiectasis, when this latter has become infected with organisms of putrefaction, in which event the condition may have become converted into one of gangrene.

(1) Fœtid bronchitis is distinguished by (*a*) the absence of shreds of pulmonary tissue and hæmatoidin crystals from the sputa, (*b*) chronicity, and (*c*) absence of demonstrable cavity.

(2) Bronchiectasis. (*a*) Under ordinary circumstances the sputum is free from evidences of lung destruction. (*b*) The expectoration is not so fœtid as in gangrene and does not contain crystals of hæmatoidin. (*c*) Constitutional symptoms are not so markedly those of sepsis. (*d*) Signs of cavity may not be definite or they point to more than one cavity. For additional particulars the reader is referred to the appropriate chapter.

Prognosis.—Both affections are of great gravity but of the two, gangrene is the more serious. Abscess may heal spontaneously when the area stands in such connection with a bronchus as to favor its free drainage. Much time may be required, but after cessation of the discharge cicatrization and contraction are possible and if the cavity is small it may eventually become obliterated. Such a fortunate termination is not uncommon in abscess from pneumonia or a foreign body.

Pulmonary gangrene is more grave because of the liability of the necrotic process to extend, even when communication with the bronchi is free. When the putrefactive area is shut off so as not to be evacuated *per os*, or when the process is multiple, the disease is very apt to result in the death of the individual. Spontaneous recovery is unlikely in any case and hence the outlook depends upon the possibility of surgical interference.

Either affection, when of embolic origin, especially if multiple, is very likely to terminate fatally, in some instances the result being due to the infarcts themselves, in others to the original affection. Complications, as septic pleuritis or brain abscess resulting from the pulmonary disorder, add to the danger and may preclude all hope of cure either by the surgeon or by nature.

The COURSE of either affection, but particularly of gangrene, may occasionally prove very rapid, death occurring in from several days to as many weeks. Nevertheless, cases are encountered which become chronic and then last for months. This is not likely, however, on account of the exhausting effects of prolonged suppuration and toxæmia.

Treatment.—From a therapeutic standpoint, cases of pulmonary abscess and gangrene may be divided into two great groups: (1) those that require surgical intervention, and (2) those which, not necessitating or not being amenable to operation, must depend upon medicinal management. For the sake of convenience these two modes of therapy will be considered in the order mentioned.

Pneumotomy.—The results of surgical treatment in pulmonary abscess and, for that matter, in gangrene also are so encouraging that no surgeon would now think of refusing to operate in any case furnishing suitable indications. Among these are the following: (*a*) If either abscess or gangrene has been diagnosed and, after a reasonable time, has not been discharged through the bronchi, the services of a surgeon should be invoked. The only question arising in such a case is, how long ought one to wait before calling on a surgeon. No exact length of time can be stated; the condition of the patient must determine largely. A waiting policy is far more dangerous in putrefaction than in suppuration.

(*b*) Should a given case of abscess manifest signs of betterment after evacuation of the necrosed tissue by mouth, operative interference may safely be deferred until the physician becomes convinced that the abscess cavity cannot heal without the aid of the surgeon or until the improvement on the part of the patient comes to a standstill, or changes for the worse. As regards gangrene, however, conditions are different. Here the necrosis may proceed despite comparatively free expectoration; so that if fœtid sputa persist and from time to time show fragments of tissue, an operation should not be postponed.

(*c*) The accessibility of the cavity or the certainty of its exact location is one of the most difficult problems confronting the diagnostician. By physical examination alone this may be absolutely impossible, and hence in every dubious case the X-ray should be resorted to.

Exploratory puncture for this purpose is not advisable, and indeed is condemned by Fraenkel for the following reasons: (1) The needle may penetrate a bronchus containing pus or putrid material and thus mislead the operator. (2) The cavity may be empty at the time and the needle fail to demonstrate the presence of the vomica. (3) A blood-vessel of considerable size may be pierced and dangerous hæmorrhage result as in a case reported by Kohn. (4) If the overlying pleural surfaces are not adherent the withdrawal of the needle may lead to infection of the sac or of the external parietal tissues. Accordingly, pneumotomy should not be decided upon until the exact situation of the cavity has been ascertained by repeated and painstaking examination.

(*d*) The ætiological character of the abscess or gangrene is also of considerable moment, since Tuffier's statistics show that the results depend in large measure thereon.

GANGRENE CAUSED BY	Recovery.	Death.	Improvement.	Nos. of cases.
Pneumonia	39	15	1	55
Bronchiectasis	1	3	..	4
Foreign bodies	1	1	..	2
Emboli	2	5	..	7
Trauma	1	..	..	1
Perforation of the œsophagus	..	2	..	2
Total	44	26	1	71

(*e*) The existence of complications. It goes without saying that certain complications, whether these be attributable to the pulmonary disease itself or are such as are constituted by the preëxisting malady, may render pneumotomy useless and hence inadmissible. Thus, one would not think of operating upon the lung if the abscess were secondary to some other obscur or perchance inoperable infectious process. Neither would an operation justifiable in the presence of a brain abscess which had led to hemiplegia or attacks of Jacksonian epilepsy, since the cerebral lesion would of itself be likely to cause death. Accordingly, in every instance with an obscure ætiology this latter point should receive careful attention before operative removal of the lung condition is undertaken.

On the other hand, the occurrence of rupture of a gangrenous focus into the pleural cavity should call for surgical treatment at once. The resulting putrid pleurisy, if not the shock, is so grave a matter that death is sure to ensue unless relief can be afforded by the surgeon. In the event of such a pleuritis the rib-resection would afford easy access not only to the pleural cavity but likewise to the gangrenous area within the lung and adequate drainage of the same.

(2) **Medicinal Management.**—This may be considered under two heads: supporting measures and remedies addressed to the healing or improvement of the local mischief. Under the former are included the nourishment, tonics, change of climate and all other measures that would naturally occur to the intelligent practitioner.

So long as the fever is high and digestion is correspondingly feeble the nourishment should be liquid or semiliquid. It must be as sustaining as possible and hence include eggs, milk, broths, meat juice, and alcohol in form of wine or whisky, but not in large amounts.

During the height of the disease the tonics indicated are strychnine, caffeine, digitalis and perhaps arsenic. As soon as convalescence has begun these may be augmented by iron, hypophosphites, cod-liver oil and such other remedies as the experience or judgment of the medical attendant may dictate.

Change of climate will be especially advisable at this time, provided the weather at home is inclement or any wise not adapted to the passing of many hours each day out of doors. Even during the acme of the illness as much fresh, cool air as the sufferer can endure should be supplied through open windows. We have, unfortunately, no more rational means of combating infection than by pure air and nourishment. We are limited to attempts to increase the resistance of the organism, and therefore, if the season is mild, the patient may be given the open-air treatment the same as the reader will find described in the Treatment of Pulmonary Tuberculosis.

Expectorants are worse than useless, in my opinion, since they are likely to derange the stomach and interfere with the patient's ability to take and assimilate food. Antiseptic remedies administered internally are likewise of very doubtful utility, and yet some of them receive the indorsement of writers. Thus Fraenkel recommends myrtol in capsules each containing 0.15 cm. (grains ij) and this dose from three to nine times daily. Creosote or guaiacol may also be ordered in suitable cases. These cases are especially such as display symptoms of sepsis. Accordingly, they are chronic cases, in particular those of gangrene, which are not amenable to surgical measures.

In chronic gangrene, also, German authors as Traube and even Fraenkel and Leyden highly indorse inhalations of oil of turpentine or rather prolonged sojourn in an atmosphere charged with its vapors. A 2 to 3 per cent aqueous solution of the oil is placed over an alcohol lamp and made to boil in a small closed apartment in which the patient remains for weeks In other cases a respirator or mask is worn over the nose and mou sponge of the respirator is kept moistened with a solution of equ creosote and alcohol. Such a device must be worn continuousl long a time as possible each day.

The fœtor of breath and sputa grows gradually less and ma serted, disappear altogether. Recovery from chronic gangrene h served during a course of such antiseptic inhalations.

Nebulized solutions of these and other antiseptic remedies ma a trial, but if much effect is to be produced the drugs must be in concentrated solutions as can be tolerated and for hours together. continuous sojourn in an atmosphere strongly impregnated is pro effective. Nevertheless Chaplin's method of inhaling fumes of cre was described in the treatment of bronchiectasis might be tried clusion, it may be added that no uncertain plan of medicinal treatn be advocated as against the opening and draining of abscess or cavities when, by skillful physical examination and the X-ray found operable.

CHAPTER XX

CHRONIC INTERSTITIAL PNEUMONIA—FIBROSIS OF THE LUNG—CIRRHOSIS OF THE LUNG—FIBROID PHTHISIS

By the above terms are indicated an induration and shrinkage of a part or the whole of a lung, resulting from an overgrowth and retraction of its connective tissue. This condition was recognized by Morgagni, Avenbrugger and Bayle, and was described by Laennec in connection with bronchiectatic and phthisical cavities. Sir Dominick Corrigan gave it the name of Cirrhosis, which is objected to by some as implying an independent condition analogous to cirrhosis of the liver. Indeed it is this very question of its pathogenesis which has engaged the special attention of pathologists and which, as yet, is not entirely settled.

Ætiology.—Although some observers, particularly among the English, have considered this condition as in many instances a part of a general tendency to fibroid change, it seems to be regarded by most writers as a result of previous inflammation or of some other antecedent affection, the precise nature of which cannot always be determined. We have, therefore, to consider its connection with the following diseases.

(1) *Acute Fibrinous Pneumonia.*—There appears to be no doubt of the possibility of an interstitial induration resulting from an acute pneumonia. It is exceedingly infrequent, and the query arises what influences determine the organization of the exudate and the development of connective tissue instead of resolution (Fig. 36 and see Plate VII). By some authors these have been found in a depraved or lowered state of the system which favored a slow reaction and tendency to subacute or chronic inflammation. Others, on the contrary, believe this interstitial thickening is not a manifestation at all of such low grade of inflammation, but is the result of repeated attacks of pneumonia in that part of the lung. They point out that this change is not uncommon in persons who give a history of repeated attacks of croupous inflammation of the particular lobe in which induration of this kind is found.

(2) *Broncho-pneumonia.*—Limited or scattered areas of fibrosis are not infrequent in children who have been the subjects of lobular pneumonia, and since, as insisted on by Delafield, a proliferative thickening of the walls of the alveoli and bronchioles is the essential characteristic of this form of pneumonia, it is not singular that permanent induration of such inflammatory areas should result. It is in secondary broncho-pneumonias that such interstitial thickening is most likely to occur.

(3) *Tuberculosis.*—Bayle appears to have first dwelt on the relation of some cases of chronic induration of the lung to tuberculosis and, since his time, all pathologists have discussed this relationship. The development of fibrous tissue is one of the steps in the tuberculous process in the lungs and seems to be a mode of healing adopted by nature (Fig. 36). This tendency is more marked in some cases than in others, being especially so in such as display a tendency to chronicity. Consequently many cases come to autopsy in which extensive fibroid changes are discovered at one apex, or possibly in both lungs. There may be bronchiectases or phthisical vomicæ surrounded by dense bands of interstitial tissue, or there may be scattered tuberculous or cheesy nodules. We cannot definitely account for this tendency to fibrosis in pulmonary tuberculosis, but must accept it as a constant feature of the disease. So constant, in fact, that authors like West and Fox seem to think it is tubercle that is responsible for every case of chronic interstitial pneumonia.

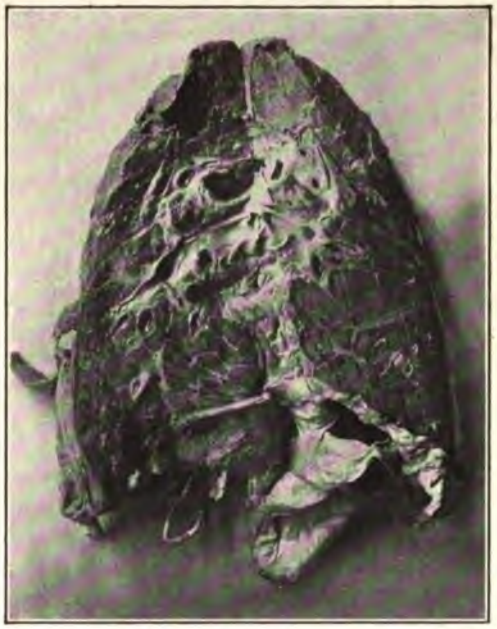


FIG. 36.—Fibrosis of lung following pneumonia.

(4) *Pleurisy.*—There is a form of very extensive fibrosis which seems to have its starting point in a proliferative pleurisy, and hence Charcot gave to such cases the term of pleurogenous induration. The process seems to originate in an inflammation of the pleura which not only leads to great thickening of this membrane but spreads more or less deeply into the lung along the interlobular sæpta. This form of pulmonary cirrhosis leads to extreme deformity of the chest and displacement of organs.

(5) *Bronchiectasis.*—There is undoubtedly some close causal connection between bronchial dilatation and chronic interstitial pneumonia, but just what this is, no one appears able to settle. By some, the bronchiectasis is considered secondary to the shrinkage that takes place in the new connective tissue, while others, as Fraenkel, hold as more likely the view that bronchial dilatation is primary. It is suggested that the retention and stagnation of secretions within the bronchiectases favor the growth of bacteria, and that it is to their action in turn that inflammation and interstitial induration are due.

(6) *Inhalation of Dust.*—There is a form of fibroid phthisis which is observed in persons whose work subjects them to the inhalation of dust

to which is given the term Pneumonoconiosis. It is especially frequent in workers in stone and steel, since the dust particles produced in stone-cutting, knife-grinding, etc., have sharp points and edges by reason of which they are especially irritating to the lungs. In spite of this irritating property of such dust, however, there are authors who believe that in this form of phthisis it is the presence of tubercle which accounts for the fibrosis.

(7) *Bronchitis.*—West speaks of bronchitis as a cause of chronic interstitial pneumonia, especially when the bronchial irritation is due to inhalation of dust. In such cases, he says, the changes are widespread but are not uniform, being in some cases most marked at the apex, in others at the base.

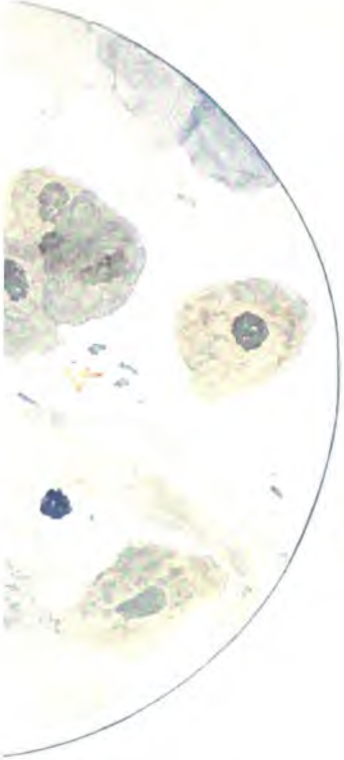
(8) *Compression by Tumors.*—It is generally recognized that when a portion of a lung is subjected to prolonged pressure, as by aortic aneurysm, mediastinal tumor, enlarged bronchial glands, etc., it may undergo induration in consequence of the development of connective tissue. I cannot ascertain that any satisfactory explanation of this tendency is known, but it is probably to be regarded as an expression of that physiological law by which, when an organ is rendered functionally incapacitated, it tends to undergo fibroid degeneration.

Morbid Anatomy.—The appearances differ only in extent and situation, not in their essential characters. Hence may be distinguished two varieties, (*a*) general or massive fibrosis, and (*b*) partial or circumscribed fibrosis.

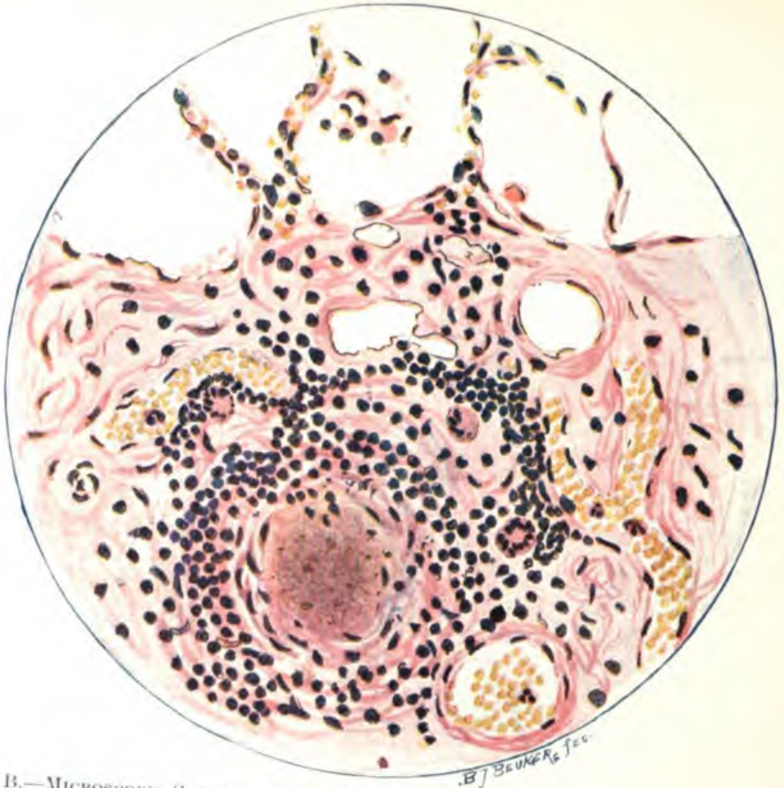
(*a*) In the general form of chronic interstitial pneumonia the entire lung is diminished in bulk, sometimes to a truly astonishing extent, is of a grayish-white color or deeply pigmented, extremely dense and hard, so that on being cut into, it creaks like cartilage. The lung may or may not be bound to the chest-wall by old, tough adhesions. In the pleurogenous variety the pleural membranes are enormously thickened and so intimately united to the lung by fibrous bands running into the lung as to render difficult the separation and removal of the organ. On section, a cirrhosed lung is found to have lost or sacrificed its normal parenchyma which has become replaced by strands or bands of fibrous tissue traversing the organ from periphery to root. The peribronchial and perivascular tissue has shared in the proliferative process as well as the interlobular connective tissue, so that alveoli and bronchioles are obliterated and their walls are thickened. Bronchiectases of varying number and size are discoverable, either empty or containing decomposing purulent material. The opposite lung is emphysematous, its anterior margin extending across the median line, and the heart is displaced toward the fibrosed lung. The liver may also be drawn upward.

(*b*) In the partial form the cirrhotic process may be limited to an upper lobe, there may be an old tuberculous focus and cavity or cavities in the midst of the dense connective-tissue bands. There may also be tubercles in the apex of the opposite lung. When this process follows broncho-pneumonia there are found areas of fibrous tissue in which alveoli and terminal bronchioles have become obliterated, and there may be bronchiectatic cavities. Such areas, separated by more or less emphysematous parenchyma, may be scattered throughout a lobe; or an entire lobe, most commonly a lower, may be seen converted into dense fibrous tissue.

PLATE VII



BERCULOSIS. TUBERCLE BACILLI
BLUE. (STAINED WITH ZIEHL'S
ALCOHOL, AND COUNTERSTAINED



B.—MICROSCOPIC SECTION OF LUNG SHOWING A YOUNG MILIARY TUBERCLE.
(STAINED WITH HÆMATOXYLIN AND EOSIN.)

If, according to Delafield, this productive inflammation follows chronic bronchitis the fibrous tissue is seen in nodules along the walls of the bronchi and more or less diffused throughout the lung, and the bronchial mucous membrane is more or less inflamed. In pneumonoconiosis the distribution of the fibrous tissue is similar to that in bronchitis, is distributed through both lungs and is deeply pigmented, particularly in workers in coal dust.

If the cirrhosis is at all extensive the right ventricle is found hypertrophied and, in some cases, other organs as well as the lungs display more or less evidence of fibrous transformation.

Symptoms.—These are essentially the same in all cases, but vary in relative intensity according to the ætiology and extent of the lesions. In all there is a tendency to chronicity, but in this respect differences are also seen.

If the disease originates in a pneumonia, the patient does not convalesce well, but gains strength slowly and is annoyed by cough, usually without more than a moderate catarrhal expectoration. Examination of the chest results in the detection of persisting dullness, bronchial or broncho-vesicular breathing and perchance a few subcrepitant râles over the seat of the previous pneumonia.

If pleurisy be the origin of the interstitial change in the lung, it may set in insidiously or suddenly with the usual characters of pleuritis. As time goes on it is found that the side does not clear up, more or less pain and cough persist, strength is regained slowly and it is discovered that the affected half of the thorax shows signs of diminution in size. In the course of months the patient's general condition improves, but more or less cough remains, the breath is a little short and the side has become decidedly retracted.

In a well-marked example in a girl of six years, seen in consultation, the history was of acute pneumonia several months earlier which had not resolved. The little patient was much emaciated, lacking in appetite and strength, coughed a good deal, especially at night, and was manifestly short of breath on exertion. The entire right half of the thorax was wanting in pulmonary resonance, being dull above and flat at the base. The respiratory murmur was bronchial, intensely so in places and of feeble intensity in others, while the voice was bronchophonic. Râles were nowhere detected. The left lung was hyperresonant. The antero-lateral aspect of the affected side showed appreciable though not marked flattening, but the heart was not appreciably drawn over. I subsequently learned that a year or two later the lung had grown somewhat more resonant but was still dull and slightly retracted. Her general condition, however, was good.

If in a given case of massive cirrhosis, the lung undergoes marked shrinkage in volume, nature attempts to compensate the retraction by a corresponding diminution in the diameter of the thorax. The ribs assume a more oblique direction, the intercostal spaces become contracted until, in pronounced cases, the rib-borders may seem to be actually contiguous. The shoulder of that side is depressed and the spinal column deviates to the same side. The liver ascends and the heart is displaced, even to the extent of producing dextrocardia when the right lung is the one at fault. In extreme examples of deformity dyspnœa may become the leading feature and the heart's action is likely to be embarrassed on exertion.

Bronchiectasis is very apt to be developed and then the symptoms are those of bronchial dilatation: cough, usually paroxysmal, purulent perhaps greenish expectoration, and even fœtor of the breath and sputa. Emaciation gradually appears, transient pyrexia is added, the function of the various viscera is disordered, strength wanes and the individual becomes a chronic invalid.

I recall the instance of a man seen in the early years of my practice whose chronic interstitial pneumonia had resulted from a pneumonia or pleurisy following trauma when a lad of twelve years. His right side was greatly flattened and retracted and the heart lay completely to the right of the sternum. His symptoms were so suggestive of chronic phthisis that the purpose of his visit was to ascertain whether or not consumption was present. This man was still alive and in somewhat better form nine years subsequently.

If such cases are not terminated by some intercurrent affection, as acute bronchitis or by an added tuberculosis, the state of the patient grows slowly worse or shows ups and downs, until at length the overtaxed right heart yields to the strain and life is terminated by cardiac incompetence.

In fibroid phthisis signs of the disease exist at one apex or possibly in both apices and the history is apt to be that of an insidious development with years of cough, expectoration, progressive emaciation, slight fever at different times and incapacity for more than very light tasks.

Examination reveals flattening of the affected apex in front, marked depression of the supraclavicular and suprascapular regions, projecting scapulæ, dullness on percussion, perhaps of tympanitic quality, bronchial breathing and creaking or dry crackling râles and voice-sounds indicative of cavities.

The aspect of the patient is that of one having some chronic disease, and the pulse is accelerated and weak. These various signs, differing in degree, depend on the extent of the fibrosis and the rapidity or slowness with which the process has advanced. If acute tuberculosis becomes added, as sometimes happens, the symptoms are those of a rapidly wasting disease with acute destruction of pulmonary tissue.

Hæmoptysis may occur in these cases provided a vessel becomes ulcerated during the destructive process. In fibroid phthisis it may result from rupture of a small pulmonary aneurysm, but such is not common. When, on the contrary, bronchiectasis has supervened and is the cause of hæmorrhage the symptom is due to a destructive process that has involved the bronchial wall and neighboring vessel.

Chronic interstitial pneumonia may display all possible degrees of difference in its clinical picture. When the fibrosis is not extensive it is not likely to affect the general health seriously and certainly does not incapacitate the individual, for years at least. When, on the contrary, it is extensive and has rendered one lung or a major portion thereof practically useless it puts a great strain on the right heart and so seriously interferes with circulation and respiration as to produce more or less chronic invalidism.

The COURSE of most cases of chronic interstitial pneumonia is essentially chronic, and unless the individual is cut off by some intercurrent affection the disease in question is likely to endure for many years. In exceptional instances a pulmonary fibrosis that has originated in acute pneumonia may lead to death after the lapse of a few months. The final breakdown in chronic cases is very apt to come from failure of heart power, such asystolism developing slowly or being the speedy result of an acute broncho-pneumonia.

Physical Signs. **Inspection.**—The portion of chest overlying the cirrhosed area appears retracted and immobile on inspiration (Fig. 37). If an entire lung, or if only a lower lobe is indurated, the respective side looks smaller than its fellow, the shoulder droops and there is scoliosis to correspond. If the cardiac impulse is visible, it is displaced toward the retracted side and the opposite lung may show compensatory development.

Palpation.—Pectoral fremitus is usually modified. It is decreased or abolished when the fibrosis is extensive and particularly when it is of the pleurogenous variety. If bronchiectatic or phthisical cavities exist, tactile fremitus is likely to be retained or even exaggerated over such areas.

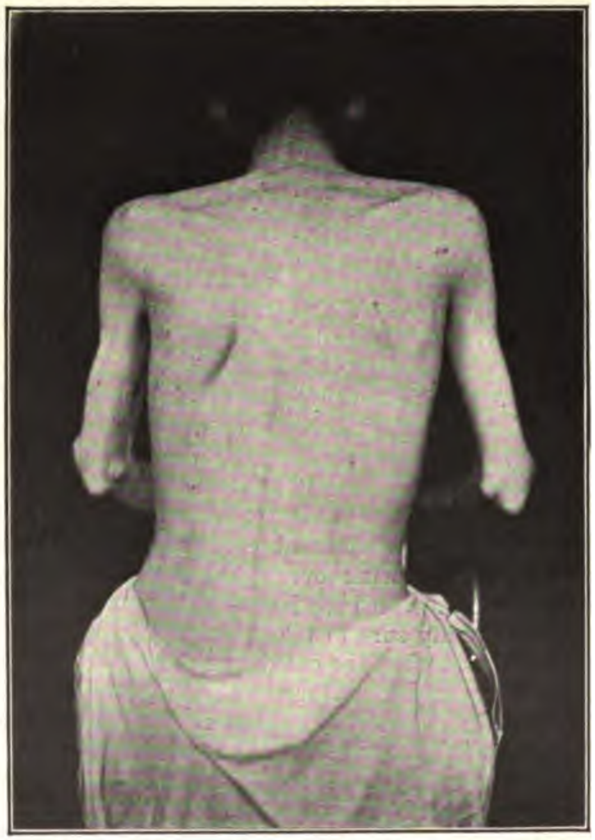


FIG. 37.—Asymmetry of chest in chronic interstitial pneumonia.

Percussion.—Pulmonary resonance is diminished or even wholly replaced by dullness that may amount even to flatness. Percussion is attended with a sense of greatly increased resistance. Cavities may occasion a high-pitched tympanitic note in certain areas in the midst of intense dullness. Such is especially the case at the apex in fibroid phthisis.

Auscultation.—The normal respiratory murmur is lost, and instead there is broncho-vesicular or tubular breathing. This may be intense, in cases of apical fibrosis, or there may be spots over which the breath-sounds are very suggestive of cavity. In pleurogenous cases the respiratory murmur may be entirely abolished. The voice is distant and feeble or bronchophonic, according-ing to the extent and density of the cirrhosis and the absence or presence of cavities. Indefinite creaking sounds may sometimes be heard especially along the margins of the lungs or where dullness merges into comparative pulmonary resonance.

Diagnosis.—The recognition of chronic interstitial pneumonia is ordinarily not a difficult matter. The history of some antecedent affection capable of leading to fibrosis, together with the combination of physical signs described above, enables one to arrive at a correct diagnosis. This is especially true of the massive form, but when areas of fibrosis are small and scattered their existence may be only a matter of conjecture.

Differential Diagnosis.—No other chronic pulmonary affection produces dullness and bronchial breathing together with diminution in the diameter of the affected side. There are cases, however, in which it is difficult to say whether or not the signs are due to pleurisy with effusion. It is possible for pleuritis to have developed in a side already smaller than its fellow in consequence of spinal curvature. In such a case, as in every other of pleuritic effusion, great aid will be obtained by carefully noting the position of the heart and other organs. Intrapleural collection of fluid displaces organs toward the sound side, whereas in chronic interstitial pneumonia their dislocation is toward the diseased side. For additional differential points the reader is referred to the chapter on pleuritis.

Cases are occasionally encountered in which it is impossible to say positively whether the flatness over the side is due to thick pleuritic adhesions without cirrhosis of the lung, or whether the condition is one of independent cirrhosis. It is generally safe to assume in these cases that the productive inflammation has invaded both the pleural cavity and the interlobular connective tissue. Permanent compression or partial collapse of a lung may result from pleuritic overgrowth and in time the chest-wall may retract somewhat. But in such instances hyperplasia of the interlobular connective tissue is pretty sure to take place, in time, and the lung to become more or less cirrhotic when the case becomes really one of the pleurogenous variety.

The most difficult question calling for answer is that of the existence or nonexistence of tuberculosis in these cases of chronic interstitial pneumonia. The answer must be found in the history and in the discovery or not of tubercle bacilli and shreds of pulmonary tissue. One should not be satisfied however with one or two negative sputum examinations, since the germ may not be discovered until after the lapse of months even, in tuberculous cases. The supervention of acute phthisis is usually not difficult of diagnosis, the rapid wasting, the signs of forming cavity and the character of the sputa making the nature of the case clear.

Prognosis.—Chronic interstitial pneumonia is an incurable disease and hence the question confronting the physician relates to the life-prospect of the patient. Nature is wonderfully tolerant at times of pathological processes, and especially so where these are chronic. Consequently, even the induration of a whole lobe or of an entire lung may be endured for many years. The individual is not likely to be capable of much work, but with a favorable environment he may still enjoy a fair measure of usefulness and happiness.

If this is true of massive pulmonary cirrhosis, then how much more it applies to limited forms of fibrosis. Children in whom it follows broncho-pneumonia may, in course of time and under good hygienic conditions, grow up into fairly robust citizens. They are, however, more susceptible to acute bronchial and pulmonary disorders than they might otherwise be.

Even fibroid phthisis is not prognostically so bad as might be thought. This form of tuberculosis is essentially chronic and therein lies its relative safety. The tuberculous zones and cavities may be walled off by dense connective tissue and thus the disease be kept from spreading. However, when persons with considerable pulmonary fibrosis fall victims to acute intercurrent disease, they are likely to manifest impaired resistance and they may readily succumb to what might under other circumstances prove a comparatively trifling ailment. When at length the chronic interstitial pneumonia has led to extensive bronchiectases, as it is claimed to do by Sir Andrew Clark and others, or when it has followed upon already existing bronchial dilatation, the prognosis is less favorable and may, in some instances, be serious, depending upon the severity of the constitutional disturbance caused by the bronchiectasis.

When grave circulatory embarrassment supervenes after years of tolerance on the part of the heart, then prognosis is exceedingly bad, for since the cause of the cardiac inadequacy is irremediable there can be no hope of completely reinstating heart power. If the compensatory emphysema of the opposite lung should become such as of itself to be a serious element in the case, prognosis is doubly grave, for there are then two conditions to contend with, each in itself bad.

If, finally, tuberculosis becomes added, as may happen, the cirrhotic tissue, being poor in blood-vessels, may undergo rapid destruction (Fraenkel). The course of the disease then becomes acute, cavities form rapidly and death may supervene in a few weeks. The case has then become converted into an acute ulcerative phthisis.

Treatment.—Chronic interstitial pneumonia is not amenable to therapeutic measures, or, in other words, we possess no medicinal or other means capable of restoring a fibrous lung to a normal state. Therapy must be limited, therefore, to attempts to modify symptoms and to keep the general health in as good condition as possible. In a considerable number of cases, after the cirrhosis has become fully established, the patient's health is such that medication is not required and medical services are not often sought.

If the individual is a child who has recovered from broncho-pneumonia, the family doctor should keep a general oversight of the case and promptly treat those intercurrent ailments to which such a child is liable. It may even be well to send him away into a suitable climate for the winter or at such times as the little one does not appear to thrive.

Cough or dyspnœa are the symptoms most likely to call for medical aid. If the former prove especially troublesome, the best measure would be a change of residence to a suitable climate whenever this is possible. Should such be impracticable, then the symptom will have to be treated by inhalations, e pectorants and sedatives the same as in any other pulmonary complaint.

child will probably improve in a gratifying manner on cod-liver oil, iodide of iron and hypophosphites. In all cases, however, it is most important to keep the child out of doors as much as possible and to keep up good nutrition.

Dyspnœa, if due to extensive fibrosis, cannot be wholly controlled, and yet, much of the shortness of breath may be overcome by carefully conducted breathing exercises since they tend to the development of compensatory emphysema of the opposite lung as well as the healthy parts of the cirrhosed lung. As a matter of fact, nature grows very tolerant of chronic disorders provided they are not of the kind to impair health by absorption of toxic products or to interfere seriously with processes of assimilation and growth.

Cases showing signs of bronchiectasis must be managed symptomatically along the lines laid down in the chapter devoted to that disease. Such cases do best in a mild, dry climate where the days can be passed out of doors. Patients whose means necessitate their remaining in their home climate, no matter how adverse this may be, must be content with such therapeutic measures as will ameliorate their most troublesome symptoms.

The development of pulmonary tuberculosis necessitates management along the lines laid down for that disease but with such modifications as are required by the fibrosis. If the process is a rapidly destructive one, it is folly to send the patient from home but if chronic it may be improved by permanent removal to some health resort of low or comparatively low altitude. Especial pains must be taken to maintain nutrition and the cases must be managed as would any case of pulmonary tuberculosis.

The development of cardiac inadequacy offers an indication for heart tonics and for more or less complete physical rest, the same as if the incompetence were of other origin. The probability is that the dilatation of the right heart cannot be wholly overcome, and yet, skillful treatment may at length reëstablish a fair measure of hypertrophy.

CHAPTER XXI

PNEUMONOCONIOSIS

Historical.—This is a general term proposed by Zenker to designate the changes in the lungs following the prolonged inhalation of dust. At the time when this subject was exciting widespread interest, and investigators were studying the effects of the inhalation of various kinds of dust-particles there were other terms in use. Thus, anthracosis was the appellation given by Stratton to Coal Miners' Phthisis, while siderosis designated the disease due to iron dust, and chalicosis that form of lung disease found among workers in stone dust. There is now no need of such distinctions since we have learned that whatever the kind of dust respired, its effect on the lung, when injurious, is essentially the same. Zenker's term is preferable, therefore, since it recognizes the pathological unity of the changes resulting from causes that differ in kind but not in effects.

It is said that even as early as 1729 Wepler directed attention to the great prevalence of pulmonary disease among French makers of millstones, and that in 1775 Le Blanc confirmed previous reports concerning the mortality among such workers. The great stimulus to interest in the subject is to be dated, however, from the writings of Pearson in England in 1813, Gregory in 1831, Stratton in 1837 and many others in England and on the Continent, among whom may be mentioned Christison, Greenhow, Peacock, Virchow, Zenker, Traube, Goldheim, Lewin, Rosenthal, Knauff, Grawitz, Arnold and many others.

Earlier observers had been struck by the black color of the lungs and of the expectoration seen in coal miners; they believed it due to the deposit in the lungs of particles of carbon, and both Christison and Peacock proved by the use of hydrochloric acid that workers in mineral dust actually took demonstrable quantities of such stony particles into their lungs. Virchow, however, threw a bomb into their ranks by declaring that the pigmentation of the lungs, seen in miners, was not caused by carbon but by a pigment derived from the blood. This led to many experiments, animals being compelled to live for days in an atmosphere impregnated with various kinds of dust. These proved that fine particles of carbonaceous, metallic and mineral substances are capable of penetrating not only the bronchi, but even the distant alveoli, in such numbers as to be demonstrable by appropriate chemical tests and by the microscope.

In 1864 Traube reported the case of a man who had worked for many years in charcoal, and at whose death clearly recognizable particles of charcoal were found in his lungs. In consequence of all this mass of incontrovertible evidence, Virchow at length, in 1866, acknowledged his error and announced his conviction that the penetration of finely divided particles into the deeper parts of the pulmonary parenchyma was not only possible but actually the case. Zenker's celebrated case of a worker whose duties required him to rub powdered oxide of iron into tissue paper, and from whose lungs the same iron salt was recovered after death, attested to the fact that other forms of dust than that from coal might be responsible for pulmonary disease. It was this observation that led Zenker to suggest the name *siderosis pulmonum* for such cases.

It is interesting, furthermore, to record that Arnold identified particles of *lapis lazuli* from the lungs of workers in this blue pigment, and various silicates from the lungs of stonecutters. Along this line also are the significant investigations of Kussmaul and Schmidt to determine whether or not silicic acid can be discovered in the pulmonary tissue of healthy organs. Whereas a two-weeks'-old infant showed not a trace of this substance in its lungs, the percentage of ash was found to exist in increasing amounts with advancing years in the lungs of older individuals, ranging from 4 to 17 per cent in adults and reaching the astonishing figure of 24.7 per cent in a stonecutter.

Accordingly, Kussmaul gives 1 gramme as the average amount of silica present in the lungs of persons who do not work in stone dust, while in a worker in such an atmosphere it may reach 3 grammes. Meinel corroborated these results, finding 30.7 per cent of this substance in the ashes of the lungs of a glasspolisher, and in another workman, who died of pulmonary tuberculosis, the enormous sum of 45.6 per cent (Fraenkel). It was this last-mentioned investigator who suggested the name *chalicosis* for this class of cases.

These and many other researches and observations established beyond question the possibility of pulmonary disease resulting from the inhalation of dust. They also showed that all forms of dust are not equally hurtful. Thus, vegetable particles are by no means so injurious as those of metallic or mineral origin owing to the sharpness and hardness of these latter. Accordingly, some trades are far more harmful than others, namely, needle-pointing, knife-grinding, diamond-cutting and, in short, all such as create a mixed metallic and mineral dust.

The recognition of the above facts led to the adoption of means calculated properly to ventilate workshops or to precipitate the dust (usually by moisture) and thus prevent its dissemination through the air of the room. Consequently, this disease no longer obtains in such proportions as during the fore and middle part of the last century. In the United States pneumonoconiosis is a rare affection and, outside of mining regions and large manufacturing centers, is scarcely if ever encountered. I certainly have never seen a case that may properly come under this head. I recall the instance of a coal miner, a young man of less than twenty, whose attending physician brought him to me on the supposition of his having Coal Miners' Phthisis, but who was suffering from a plain case of pulmonary tuberculosis, and his expectoration was not appreciably stained with carbon particles.

Ætiology.—This has been practically covered in the foregoing introductory remarks. The fact of the inhalation of dust and of its penetration into the deeper structures of the lungs has been sufficiently attested. The real question confronting us in considering the causation of pneumonoconiosis is, therefore, whether dust particles are in reality as irritating and injurious as was once supposed, or whether the destructive changes discovered *post mortem* are to be attributed to the dust or to the presence of tubercle bacilli.

Observers seem pretty well agreed that coal dust and that arising in the reduction of limestone to chalk is not of itself especially hurtful. The lungs become pigmented and the expectoration is blackish, but consumption is comparatively infrequent among coal miners, excepting among such as labor in the deeper galleries where they have to breathe the fumes of gunpowder used in blasting out the coal as well as the smoke from their oil lamps (Fox). Halter and Grab are said by Fraenkel to have regarded chalk dust as likewise not especially deleterious, an opinion also entertained by Sommerfeld as respects the dust from plaster of Paris. They actually thought such dust might exert a somewhat protective influence against the development of pulmonary tuberculosis.

On the other hand, all forms of dust made up of sharp, hard particles exert so pronounced an irritation that serious disease ensues, either chronic bronchitis or tuberculosis. Consequently, workers in charcoal, grinders of all kinds, stonecutters, workers in copper and pottery are particularly liable to suffer from the disease now under consideration. The same holds true of persons who inhale organic dust, especially tobacco, while certain of the operatives in cotton factories, namely, mixers and batters, are said by Wilson Fox to suffer the most.

The introduction of more sanitary surroundings, i. e., larger workrooms and systems for securing adequate ventilation, have materially lessened the prevalence of pneumonoconiosis. Not only is less dust inhaled, but the general character of the atmosphere is improved, and hence the workmen are better able to withstand the harmful effects of their occupations. In the case of many, notably cigarmakers, it is quite reasonable to assume that the deleterious influence of dust, tobacco, e. g., is intensified by their posture while at work. They sit all day bent over in such a manner as to restrict respiratory movement and hence retard the expulsion of the foreign particles that have gained access to the trachea and large tubes. In this connection it may be stated that, thanks to the intelligent efforts of the Cigarmakers' Union in obtaining better ventilation, the death rate from consumption among its members has been materially reduced. In the case of metallic and mineral dust we do not need to look for such accessory factors since, if inhaled in sufficient quantities, it is directly and powerfully irritating to the pulmonary tissues.

A more interesting query regards the relationship existing between pneumonoconiosis and tuberculosis. Before the work of Koch had furnished reliable data for the recognition of tubercle, observers interested in this phase of the subject were compelled to rely on the gross and microscopic appearances without being able to recognize the bacilli. It was natural, therefore, that the cases of phthisis seen in persons exposed to inhalation of irritating dust should have been regarded as ætiologically different from ordinary consumption. Still, even at that time, there were some who inclined to the opinion that the inhalation of dust only predisposed to tuberculosis, and that, apart from the presence of tubercle, destructive changes were not likely to occur.

At the present time, however, this subject cannot be said to be fully cleared up. In the lungs of pneumonoconiotic patients there are nodules and fibrous masses whose exact nature is not conclusively established. Some observers still appear to hold a conservative opinion regarding them, while others look upon them as tuberculous. This latter view seems strengthened by the discovery of tubercles on the pleura or peritonæum of some subjects whose lungs appeared to furnish evidence of pneumonoconiosis only.

Fowler and Fraenkel hold very positively that the inhalation of dust in any considerable amount must be looked upon as one of the factors *predisposing* to pulmonary tuberculosis. In the light of modern pathological and bacteriological knowledge I do not very well see how one can hold to any other conviction than that the dust, as such, may lead to chronic bronchitis and to slow indurative processes, whereas the breaking down of lung results from the action of the tubercle bacillus, either secondarily or, as held by some, primarily, the tuberculosis favoring the injurious action of the dust.

Sommerfeld's tables given by Fraenkel prove beyond question that pulmonary tuberculosis is more common among individuals exposed to a dusty atmosphere than among persons not so exposed. The influence of dust in this direction may be attributed to irritation of the bronchial mucosa and injury to its protective epithelium, but Fraenkel thinks it far more likely that the tuberculosis may be referred to obstruction and obliteration of the lymph channels. In those instances in which pulmonary tuberculosis is not produced the bacilli are carried by the lymph stream to neighboring bronchial glands or to other distant organs where they undergo destruction. Whatever be the ultimate settlement of such questions, the discovery of bacilli in the expectoration of this class of patients proves that a special form of phthisis due exclusively to dust does not exist, but that workers in a dust-laden atmosphere are especially liable to consumption, this tendency being enhanced by their unfavorable home environment, their individual habits, resistance, etc.

Morbid Anatomy.—The changes in the lung induced by the inhalation of the various kinds of dust are essentially the same, the chief points of difference consisting in the color, and hence, for the sake of convenience, the various forms of disease, anthracosis, siderosis and chalicosis, may be described together. The degree of change in each depends largely upon the quantity of dust that has been taken in, the length of time during which the lungs have been subjected to dust-inhalation, and upon the irritating quality of the particles. Other factors also coming into play are individual resistance and the supervention of tuberculosis.

The first thing that attracts attention is the color of the lungs. In anthracosis the organs are dark or even black, while in siderosis they are dish hue and in chalicosis they are of a gray appearance. Other kinds

of dust stain the lungs of a hue corresponding to the color of th
peculiar to the mineral. Thus workers in ultra-marine may hav
a distinctly blue appearance. The color, whatever it may be, is
ficial but invades the entire organ.

On incising the lung of a person who has suffered from a
e. g., the cut surface is smooth, often densely black, and the s
flows may look like black ink. The tissue is dense and hard from th
ment of fibrous tissue either in nodules or in large masses. Bet
areas the lung is emphysematous, while the bronchi display the
and catarrhal swelling of chronic bronchitis or are more or le
The fibrous tissue cuts with difficulty, and in chalicosis may grate
knife from the gritty nature of the particles contained in the
tissue.

These areas of induration may be scattered throughout the org
greatly preponderate in one or another lobe. Thus, in one case
be in the central portion of the lung a mass of the size of an oran
larger, while in another the upper lobe may show the characteristi
In some instances this newly formed connective tissue may have
necrosis with consequent formation of an irregular cavity with ra
Such destructive change is probably the result of tubercle, altho
appears to think it may take place without such intervention.

The manner in which these various changes take place is the
The dust is first taken into the trachea and bronchi where, en
mucus cells it is, through the defensive action of the ciliated e
cast out in the expectoration. When, however, it is too abundant
gotten rid of, or when the protecting epithelium is shed in places
enters the mucosa and penetrates into the lymph spaces. Here i
upon by the connective-tissue cells and is disposed of.

Should the dust-particles be too abundant to be successfully tak
by these cells they enter the lymph stream and are carried to m
parts, i. e., to the lymph nodes surrounding the bronchi and bl
to the interlobular connective tissue beneath the pleura, and to th
and mediastinal lymph glands where they are retained and thus
from entering the general circulation (Arnold). This attempt o
of nature to dispose of these irritating foreign particles has its limit
and hence, in time, there is set up the sclerosis of connective ti
results in extensive areas of fibrosis.

Such changes alone would not prove especially serious, and s
toms as might exist would probably result from the associated
The resistance of the pulmonary tissues is impaired, however,
time or coincidently, tubercle bacilli are also inhaled, destructive c
induced which result in excavation. Thus it is that vomicæ ar
which in no wise differ from those seen in ordinary consumption.

Symptoms.—These are determined by the nature and extent
dominating changes in the lungs. For a long time, years it may
in a dust-laden atmosphere may follow their occupation without e
any special inconvenience. At length, however, *cough* sets in a

as attends chronic bronchitis. It is attended with expectoration of mucus usually in small masses or lumps, and stained of a color corresponding to the kind of dust responsible for the cough. In anthracosis it is grayish-black or even black so as to resemble thick ink, while in siderosis the sputum is reddish-yellow or black, depending upon the kind of iron salt inhaled. Examined by aid of the microscope the sputa are seen to contain minute fragments or particles of coal, steel or silica, as the case may be, the dust-components being contained in the mucous and alveolar cells that largely make up the expectoration. In the case of stoneworkers it is said that occasionally tiny fragments of silica may be discernible by the unaided eye (Fraenkel). In Traube's case, particles of charcoal were easily recognized under the microscope.

After the bronchial catarrh has existed for a longer or shorter period *shortness of breath* is added to the symptoms. At first noticed only on exertion, or at the close of a hard coughing fit, it at length becomes wheezy or asthmatic and is then due largely to the associated emphysema. If fibrosis be extensive or the right ventricle feeble, and especially if destructive changes be great, the consequent dyspnœa may be extreme and form a marked feature of the case. By this time *emaciation* and *debility* are added and the patient seeks a hospital or takes himself to bed from weakness and inability longer to endure his distress without medical aid.

Examination of the chest may disclose only signs of bronchitis, with or without areas of dullness, and bronchial breathing corresponding to the fibrosis. In many cases the symptoms now point strongly to a phthisical process, and the microscope may reveal tubercle bacilli and even small portions of pulmonary tissue. In such an event, signs of cavity may be detected at one or the other apex, *fever* is present, loss of strength and flesh are pronounced and the case presents every appearance of ordinary consumption; indeed it would be so considered were it not for the peculiar pigmentation or grittiness of the sputum.

The COURSE of pneumonoconiosis is essentially chronic, but should tuberculosis be added to the fibrosis the downward progress of the disease becomes hastened. If the patient leaves his occupation during the stage of bronchial catarrh and seeks treatment his symptoms may be so greatly relieved that he will even consider himself cured. Such was the history of a coal miner who was an inmate of Cook County Hospital during the Summer and Fall of 1904. He sought admission because of cough, expectoration and associated symptoms due to chronic bronchitis. After a sojourn of several months his condition was so improved that he left the hospital, as he thought, cured. It is worthy of note that his expectoration, at first thick and as black as ink, gradually lost its carbon and was no longer black at the time of his discharge.

This is in accord with the observations of Merkel who, according to Fraenkel, was unable to detect the bluish pigment in the sputa of a worker in ultramarine for longer than fourteen days after he had abandoned his occupation. In Traube's first case, at the end of four months the sputa entirely lost their previously characteristic hue, although the man had labored for years in charcoal dust and the œdematous lungs must have been thoroughly impregnated with minute particles of charcoal, as evinced by the grayish-black color of the frothy serous expectoration. In such cases one is not to conclude that the lungs have gotten rid of the dust-particles which have penetrated their interlobular connective tissue, but merely that what dust was contained in the air-tubes, i. e., in the mucous and alveolar cells (dust-cells), has been removed.

Physical Signs.—There is nothing pathognomonic in these, since they are only such as are found in other diseases that lead to induration and finally to ulceration of the lungs. In the early stage, i. e., as long as chronic bronchial catarrh is the only result of dust-inhalation, examination of the chest is negative and hence distinctive signs appear only when fibrous transformation and excavation have become considerable.

Inspection perceives more or less shortness of breath and respiratory movements may be restricted on one or both sides and at apex or base, as determined by the extent and situation of the lesions. Although more or less emphysema is associated with fibrosis, the thorax is not likely to be barrel-shaped. Instead the apices are retracted and the infraclavicular regions flattened. There may also be epigastric pulsation and distended superficial veins, particularly on the arms, indicative of the right ventricle dilatation and impeded circulation through the lungs.

Palpation.—The induration of pulmonary tissue may be shown by increase of pectoral fremitus, either general or localized. If ulceration has led to formation of cavity this is likely to be apical, and hence there may be marked exaggeration of fremitus over such an area beneath one or the other clavicle.

Percussion.—So long as chronic bronchitis alone exists, the percussion-note is resonant. When, on the other hand, induration supervenes, resonance becomes impaired and in some areas the note may be positively dull. Should excavation have taken place this may be shown by tympanitic resonance over a limited zone, usually in one or the other infraclavicular region.

Auscultation.—At first the breath-sounds may not be materially altered, or they may be intensified and accompanied by coarse mucous or sonorous and sibilant râles, in short, the respiratory phenomena of chronic bronchitis. Later on, in the stage of fibrosis, breath-sounds are likely to be bronchial or semibronchial depending on the extent of induration. When the breaking down of lung tissue takes place the respiratory murmur becomes distinctly tubular or even cavernous or amphoric over the area occupied by cavity. Voice-sounds are intensified and even pectoriloquous or cavernous over the same zone, and coarse clicks or even metallic tinkle may be heard, depending on conditions residing in the cavity. For signs distinctive of vomica the reader is referred to the chapter dealing with pulmonary tuberculosis.

Diagnosis.—The recognition of pneumonoconiosis ought not to be a difficult matter, especially when pulmonary changes have become marked. In the anthracotic form the inklike sputum may of itself establish the diagnosis, and if, in addition, the signs of chronic bronchitis or of consolidation and excavation be present, there can be no doubt of the existence of Coal Miners' Phthisis. In certain other occupations also, the color of the expectoration and the history of working for a long time in an atmosphere laden with dust of the same color, e. g., ultramarine, furnish sufficient data for a diagnosis of the complaint. But a blackish discoloration of the sputa alone is not to be always relied on, since dwellers in large cities often spit up mucus stained with carbon or dust simply because they daily breathe an atmosphere impregnated with smoke.

Neither is a diagnosis to be based on the history of a patient's having been a stonecutter or having worked in a dusty atmosphere, since nowadays better ventilation of workshops has largely done away with the injury that formerly prevailed. Even if signs of phthisis are present in such a worker, the physician should exercise caution in declaring the disease due to inhalation of dust. In all such cases reliance should be placed on the results of microscopic inspection of the sputa. The detection of particles of stone or steel settles the point beyond doubt. Removal from the injurious atmosphere may cause a disappearance of dust-particles from the expectoration after a time, particularly if bronchial catarrh alone exist, but when destructive lesions have resulted, it is likely that the microscope will still detect particles of mineral or iron, as the case may be. When such evidence is not forthcoming I think the nature of the case may be considered open to doubt.

Prognosis.—This is determined by the stage of the process. Simple chronic bronchitis may be relieved by permanent withdrawal from the harmful occupation. When, however, fibrosis and emphysema exist a restoration to health is impossible, and when ulceration has been added, the prognosis becomes bad. Proper management may lessen symptoms or even retard progress, but permanent arrest of the malady is out of the question.

Treatment.—This may be divided into two heads, *prophylactic* and *symptomatic*. So soon as signs of bronchial irritation appear the patient should be advised to abandon his vocation at once. He should be removed to a pure atmosphere and should be surrounded with all those hygienic conditions which minister to health and to the mitigation of his complaint. In a word, he should so live as to prevent further changes.

When graver symptoms and examination of the chest tell of the existence of unmistakable pneumonoconiosis the management of the case must be along the lines laid down for the treatment of the lesions, as found in the chapters devoted to chronic bronchitis, emphysema fibrosis, and pulmonary tuberculosis, and to these the reader is referred. The ultimate changes in the lungs either directly induced by dust or following thereon, present no new indications. They are irremovable, and the problem confronting the practitioner is how best to build up the patient's resistance and most effectively ameliorate such symptoms as occasion greatest distress. All such measures will be found amply described elsewhere in this work and need not be repeated here.

CHAPTER XXII

PULMONARY EMPHYSEMA

THE term emphysema is not altogether satisfactory since it signifies an inflation with air and is used to designate several quite widely different conditions. In applying this term to the lungs we must distinguish two chief forms of emphysema, viz., the vesicular and the interstitial, of which the former is the one to be mainly considered in this chapter.

Interstitial emphysema may be dismissed with a few paragraphs, since, by itself, it rarely forms a distinct clinical entity. It consists in the infiltration with air of the interstitial or interlobular connective tissue in consequence of the rupture of alveoli. It takes place most commonly along the margins of the lungs and is shown by circumscribed collections of air which project like blebs or bullæ above the surface of the surrounding parenchyma.

The causes of this variety of emphysema are both pathological and mechanical, the former being such as, under increased expiratory strain, favor the rupture of the alveolar walls. They are enumerated by Hoffmann as follows: (1) Trauma, (*a*) external, as penetrating wounds or contusions with resulting fracture of a rib, or (*b*) internal, from the swallowing of a needle or the like, with penetration of the gullet and contiguous lung. (2) Too violent inflation of the lungs in attempts to revive drowned or asphyxiated persons, very rare. (3) Foreign bodies in the bronchi. (4) Morbid products in the air-passages, (*a*) intratracheal tumors, (*b*) false membrane in diphtheria. (5) External violence as attempts at strangulation. (6) Involuntary or cramplike closure of the glottis, (*a*) during childbirth, (*b*) spasm of the glottis, (*c*) violent fits of coughing. (7) Attempts at exaggerated inspiration. (8) Pathological processes which lead to pulmonary rupture, (*a*) ulceration, (*b*) phthisis, (*c*) abscess or gangrene, (*d*) tumors, (*e*) cheesy lymph nodes. It is evident that of these, those coming under the last classification are the most frequent, and that some of the others probably presuppose an underlying morbid anatomical condition to render increased intrabronchial air-pressure capable of producing interstitial emphysema.

As already remarked this form of emphysema does not ordinarily give rise to symptoms either subjective or objective by which its diagnosis may be made. When, as sometimes happens, the pleura is also ruptured and air escapes into the pleural cavity, symptoms of pneumothorax develop acutely and may be very distressing (see Pneumothorax). Even when in childbirth or from other forms of violence, interlobular emphysema is produced, it presence is only declared by the associated subcutaneous emphysema which spreads over the neck and chest. In the chronic form subjective symptoms are wanting and the disease, if shown at all during life, is manifested only by hyperresonance which may not be differentiated from that due to vesicular emphysema.

Vesicular emphysema, totally different from the interstitial form, consists in dilatation or ectasia of the air-cells with consequent increase in the volume of the lungs, and not infrequently with formation of small cavities formed by the coalescence of two or more alveoli whose walls have given way. A variety of names has been given to this condition: vesicular or alveolar emphysema, in contradistinction to the interstitial form, substantive or idiopathic emphysema which Fraenkel would limit to cases showing alveolar rupture, large-lunged emphysema, in contrast to the senile or atrophic variety, and, among the Germans, Einfache Lungenblähung, or increased pulmonary volume.

Acute vesicular emphysema, to which von Basch has given the name of Lungenschwellung und Lungenstarrheit, is sometimes met with in the course of widespread capillary bronchitis. It is shown by increased volume of the lungs, exaggerated pulmonary resonance, and numerous fine râles with prolongation of the expiratory murmur.

I saw such a case in Cook County Hospital on October 28th, 1903. A girl of eighteen was brought in breathing rapidly and laboriously, unable to speak, cyanotic and cold. The history obtainable was very meager: three weeks earlier she had been taken ill with tonsillitis and was very ill for a few days, had then improved and gotten up, but remained so weak that after a few days more she again took to her bed. One week prior to her admission she had begun to show great dyspnœa.

On examination her pulse was found to be extremely feeble and rapid, the heart-sounds were almost inaudible because of the multitude of loud crackling râles throughout the chest, and obscuring the breath-sounds. The percussion-note was everywhere of a loud drumlike character, so that the area of cardiac dullness could not be determined. Her temperature was not elevated, cough was infrequent and weak, but after the ingestion of an ounce of whisky a little muco-purulent sputum was expectorated with difficulty. The condition was believed to be a capillary bronchitis with areas of bronchopneumonia which latter were obscured by acute distention of the lungs. In spite of the most vigorous stimulation the patient did not rally and died twenty-five hours later. No autopsy was obtained.

Compensatory or **vicarious emphysema** is a localized process which often develops when the whole or a part of one lung is rendered impervious to air from disease. The air entering with each act of inspiration is unable to penetrate the diseased area and therefore makes its way into the contiguous lobules, distending them and ultimately producing permanent dilatation. This is a physiological and conservative process by which the portion of the chest-wall overlying the useless area is kept from sinking inward. This form of emphysema may involve an entire lung when its fellow is incapacitated by cirrhosis, by collapse from a pleuritic effusion or by destruction from tuberculosis. It is very frequently encountered in a circumscribed p lung surrounding areas of atelectasis or old tuberculous foci.

As a general thing these vicariously emphysematous areas do the further changes to be described in the substantive variety, cases of long standing, atrophy of the alveolar walls takes pl tiguous vesicles become blended into minute cavities.

In **senile** or **atrophic emphysema** there is the same dilatatio cence of alveoli, but the total volume of the lung is greatly dimi to the size of a lemon, Fraenkel). The thorax becomes co contracted and its musculature wasted. There are dyspnœa chronic bronchitis, but the clinical picture is very different fro large-lunged variety. It forms a part of the general wasting in and is not amenable to treatment.

Ætiology.—In the study of the causation of vesicular empl problems are presented and hence almost innumerable views hav tained. Laennec, who first described the affection in 1826, r association with chronic bronchial catarrh, notably *catarrh sec,* believed that the emphysema was the result of increased intra pressure. In this regard all authors are in agreement with French clinician, but they are not all in accord as regards the *m* of this factor.

Laennec and the French school of to-day support the view inspiratory air-pressure which distends the alveoli, more powerf efforts being rendered necessary by the obstruction to the i occasioned by the bronchitis or other pathological conditions. T therefore, as the *inspiratory theory.*

The opponents of this theory champion what is termed t *theory.*

According to them it is not the entrance but the exit of air dered so difficult as to induce the changes seen in substantive This interference with free expiration may depend upon a vari tions, as narrowing of the lumen of the air-passages or merely t force of cough, or a combination of both these factors. It however, that if the inspiratory hypothesis holds at all, it ca to cases of compensatory emphysema in which the emphysem are contiguous to areas rendered useless by atelectasis, bronchiect etc. In such it is conceivable that the air driven in by forced might ultimately bring about alveolar dilatation. Inasmuch as i ized vicarious emphysema the affected part is only of increased does not display the atrophic changes seen in the substantive believed by some writers (West) that such emphysema is of th hypertrophy.

In support of the expiratory theory it is pointed out tha emphysema develops most early and markedly in those parts, i. and borders, which receive the least support from firm parietes. parts should be especially susceptible to overdistention becomes r ent if the mechanical effect of cough is considered.

The incentive to cough is followed by a sudden more or less powerful contraction of the expiratory muscles. This act forces the air within the lungs forcibly upward, toward the channel of exit, the larynx. Escape of the compressed air is prevented in part by the closure of the glottis, which takes place in response to the stimulus to cough. The column of air is, however, an elastic cushion and hence such portion as cannot escape through the trachea and larynx is driven backward, as it were, into the main bronchi. Since the bases of the lungs are the parts most powerfully compressed by the expiratory effort, the air forced back into the larger bronchial tubes is deflected, so to speak, and driven strongly into the apices and poorly supported lung margins.

Each expiratory effort in a coughing fit is succeeded by a sudden deep inspiration, and hence the lungs are subjected to great intra-alveolar pressure as long as the attack lasts. If, moreover, such fits of coughing are often repeated each day throughout a period of years, they can scarcely fail to occasion emphysema in a certain percentage of persons.

Nevertheless, it has to be acknowledged that all individuals with chronic bronchitis or other disorders that excite cough do not become emphysematous. According to the analysis of cases in Fraentzel's clinic, emphysema is seen in only 7 per cent and, excluding the senile form, in but 5 per cent. Consequently, some other factor than mere mechanical distention of the lungs seems necessary. Children rarely become emphysematous and, when they do, their emphysema is quickly or acutely developed in the course of some affection as measles or pertussis. It is a simple increase in the volume of the lungs and usually subsides after the specific acute disease has passed away. Such considerations as these have led to the view that, in addition to mechanical distention, whether inspiratory or expiratory, there must exist some disorder of nutrition, and hence we now come to speak of what is known as the nutritional theory.

The supporters of this theory base their conclusions on the observation that emphysema is especially liable to develop after measles, whooping-cough, pneumonia, etc., especially if persons recovered from such diseases resort too quickly or energetically to occupations which necessitate strong expiratory efforts. In substantiation of their contention they cite the case reported by Hertz of a cornet player who, not long after an attack of pneumonia, became short of breath. On inquiry it was ascertained that soon after his convalescence he returned to his instrument, and physical examination disclosed well-marked pulmonary emphysema. In this and similar cases the mechanical overdistention of the lungs is recognized, but inasmuch as exertion before his illness had not induced emphysema, it is argued that the real injury must be attributed to the pneumonia which, in some manner, impaired th elastic resilience of the alveolar walls.

The advocates of this theory also point out the fact first described b Virchow, namely, the want of pigment in emphysematous lungs as an arg ment in their favor. Pigment, which is in reality a staining by partic of carbon, is deposited in the lungs more abundantly as life advances a is therefore not present to any extent in childhood. Consequently its absen in the lungs of adults is taken to indicate the retention of the state normal to children, in other words of the feeble resistance normal to the lungs of early life. This being the case such lungs would be unusually vulnerable to influences tending to bring about emphysema.

An inherent, perhaps congenital, weakness in the vesicular wall being granted it is yet undetermined in what this defect consists. Cohnheim is of the opinion that it is a developmental defect in the elastic elements, and Eppinger is said to have made some observations which appear to confirm Cohnheim's view. At all events it may be stated that authors generally incline to the opinion that the underlying condition in emphysema is some disorder of nutrition and that this accounts for the injurious effect of the influences now to be considered.

Heredity.—There are a few instances on record of the occurrence of vesicular emphysema in several members of the same family. Thus Schnitzler saw it in three brothers at the age of thirty without obvious cause (West), and Hertz in four members of the same family. Nevertheless, such instances should not be taken as indicating anything more than a family peculiarity, in other words, a vice of nutrition peculiar to those families. They do not prove the influence of inheritance in all cases, nor in such a proportion of cases as to warrant its being considered a general ætiological factor.

Age.—This disease is unquestionably most prevalent at or after middle age, and yet it is by no means confined to this time of life. Conspicuous examples of it have been seen in children, and then, generally, as a sequel of some acute pulmonary affection, as measles or pertussis, or in connection with chronic bronchitis. In such instances the emphysema should be put down to the antecedent or associated disease and not to the age *per se*. The same remark may be made of the influence of middle age in its production. Persons who manifest emphysema at this time of life have been exposed for a long period to the influences which lead to emphysema and, with the exception of that variety known as senile emphysema which, in its nature, differs widely from the substantive form, their age cannot be said to be a direct causative factor. If it were, then the disease should be infinitely more common than it is.

Sex.—Males are strikingly more subject to emphysema than are females. This predominance in men is not, however, due to any predisposition inherent in sex itself but must be ascribed to the greater exposure of men to certain ætiological influences, as bronchial catarrh, occupations, etc.

Occupation.—If the expiratory theory is correct, then certain occupations must tend to the production of emphysema. They are such as entail abnormal and frequently repeated expiratory effort over a long period of time. Accordingly, the disease is relatively frequent in musicians who play upon wind and reed instruments, glassblowers, porters, day laborers, and all others who are obliged to lift and carry heavy burdens or who must maintain prolonged and severe expiratory effort. The fact that emphysema does not afflict all persons following such employments makes it not unreasonable to assume some additional element, i. e., an inherent nutritional weakness, or some of the diseases which will shortly be shown to possess an ætiological influence.

But whatever be the precise explanation, the relation of occupation to emphysema is too intimate to be ignored.

In Germany, where the disease appears to be more frequent than in the United States, I saw a number of cases which exemplified the influence of occupation. Yet, in this country I have observed several well-marked examples. Thus, I distinctly recall a young Swede of fifteen years of age who was a member of a brass band and who sought medical aid on account of such a degree of shortness of breath as to make it difficult for him to play his instrument. To my surprise his lungs presented well-marked signs of emphysema and he was advised to leave the band.

Another extremely typical example of large-lunged emphysema observed in the early years of my practice in Chicago was in an Irishman well on toward forty who had been a sailor. This man's chest was so round and distended that he could not cough effectively and found locomotion very difficult. There were no râles and no expectoration denoting bronchial catarrh. On this account I was at a loss to explain the production of his emphysema, since there did not seem to be anything in the life of a seafaring man that should cause his disease. His sputum was scanty and tenacious, and now, in recalling this fact, I am inclined to attach much ætiological importance to this character of the expectoration, in accordance with Hoffmann's opinion of such cases to be considered subsequently.

I also recollect having seen, in consultation with the late Dr. Charles Gilman Smith, a Jewish merchant of middle age who was confined to bed by reason of dyspnœa clearly due to his pronounced emphysema. In this case I could ascertain no cause for his pulmonary condition. It is in such cases with an absolutely negative personal history that the assumption of a nutritional defect seems the only reasonable explanation.

Chronic Bronchitis.—The association of emphysema with chronic bronchial catarrh is too common to be accidental. It was observed by Laennec and has been dwelt on by authors ever since. That great French clinician very rightly recognized the causal connection of the latter with the former but, as previously stated, believed the catarrh was operative through its impediment to inspiration. The correct explanation lies in the hindrance it occasions to the exit rather than the entrance of air. Cough may intensify the effect of embarrassed respiration but is not the main factor.

Moreover, as pointed out by Hoffmann, it is not catarrh of the larger tubes which gives rise to emphysema, but it is the presence of tenacious mucus in those cases in which the inflammation has extended to the smaller divisions of the bronchial tree. The secretions do not prevent the ingress of air, since inspiration is powerful enough to force air into the bronchioles peripheral to the seat of obstruction. It is otherwise, however, with e
tion. To its accomplishment muscular effort must aid the elastic rec
the lungs. In consequence, the alveoli become distended with each respi
act, and this internal pressure leads, in time, to their permanent expa
i. e., to diffuse vesicular emphysema.

That the real factor in the causation of this disease is expiratory
proven by the clinical observation that old asthmatics invariably becoı

physematous after years of suffering. In them we have all the requisites, chronic catarrh of the finer tubes, tenacious secretions and overdistention of the air-vesicles. In time also the right heart dilates, the pulmonary arteries grow atheromatous, and there are then added the conditions which aggravate the catarrh through congestion (the Stauungskatarrh of the Germans), as well as the condition of rigidity produced by turgescence of the pulmonary vessels and styled by von Basch "Lungenstarrheit."

Whooping-cough and **measles,** especially the former, are well-recognized predisposing causes of emphysema. There is not only the influence of powerful expiratory efforts and impeded exit of the respired air, but the acute disease seriously affects the nutrition of the lungs. Children may, and generally do, recover from the alveolar dilatation, but if the elasticity of the parenchyma is too seriously impaired, the emphysema may be permanent.

Other acute pulmonary affections, as pneumonia, do not lead to diffuse emphysema, though localized or vicarious emphysema is common during the course of the affection. It should be borne in mind, however, that an acute pulmonary disease may, by impairing the resistance of the alveolar walls, predispose to the complaint now considered if the individual too soon after convalescence return to an occupation which involves powerful and oft-repeated expiratory efforts. The case of Hertz's patient, already cited, is one in point.

Heart Disease.—Every clinician who has observed cases of emphysema has been impressed by its frequent association with cardiac disease. Most of them, like myself I think, have looked upon the association either as accidental or in the light of an effect, not a cause; in other words, they have regarded the emphysema as primary and the cardiopathy as secondary.

The cardio-vascular conditions are, for the most part, chronic arteriosclerosis, chronic myocarditis and hypertrophy with dilatation of the right ventricle. If murmurs are present they may denote valvular disease, but as a rule they are regarded as relative, or muscular.

It is interesting and suggestive, therefore, to note what Hoffmann has to say concerning the connection between emphysema and cardiac disease.

To begin with, he cites the statistics of Chambert showing the relative frequency of various cardiac lesions to emphysema as follows. Among 258 cases of this affection he found diseased,

Heart muscle alone	30 times.
Musculature and valves	58 "
Muscle diseased, aorta sclerotic	15 "
Valves alone	18 "
Aorta alone atheromatous	19 "

Hoffmann then remarks that it is quite clear that emphysema cannot occasion valvular disease although the relation of atheroma, synechia and disease of the myocardium to the pulmonary affection may be questionable. His own opinion is that they are primary, although he admits that in man cases the emphysema is partial, not general. There are, however, all possil gradations, from the circumscribed to the diffuse form, and in actual practice it is next to impossible to differentiate these two forms in every instance.

The difficulty of determining which condition is primary and which secondary is increased, since the pulmonary disease may undoubtedly lead to hypertrophy of the heart, with secondary changes due to disturbed cardiac nutrition.

Hoffmann then points out that cardiac disease may predispose to emphysema through the catarrh due to stasis and through the other effects on the elasticity and size of the lungs, to which von Basch and Grossmann have called attention. The latter, by experiments on dogs, established the fact that the position of the diaphragm and the volume of the lungs stand in direct relation to the state of the pulmonary circulation. In other words, when cardiac dyspnœa results from incompetence of the left ventricle there are descent of the diaphragm, increased intrathoracic pressure and, at the same time, an increase in the size of the lungs depending upon the stasis in the pulmonic system. On the other hand, when the flow of blood into the lungs from the right ventricle is impeded (pulmonary anæmia), an opposite state of things results: the position of the diaphragm is elevated, intrathoracic pressure sinks, and the volume of the lungs is diminished.

In the former condition the congested lungs cannot unfold normally during inspiration and hence the mechanical effect of respiration is hindered. In the anæmic lung on the contrary the respiratory play and hence the mechanical effect of respiration are increased.

In the light of these experiments Hoffmann is of the opinion that the emphysema of cardiac origin is referable to the rigidity of the lungs incident to stasis, and that this enlargement of the lungs from congestion is too often mistaken for emphysema. Yet when emphysema and pulmonary stasis coexist the former is in reality the consequence of the bronchial catarrh occasioned by the stasis. The subject is one of much interest and merits further investigation.

Climatic and social conditions, inhalation of dust, etc., are predisposing factors in emphysema only in so far as they conduce to chronic bronchitis and, through this, to the disease now under discussion and may therefore be dismissed from further consideration.

The consideration of the ætiology of emphysema would not be complete without mention of the theory advanced by Freund which, if not generally accepted, yet possesses the merit of originality. I cannot, therefore, agree with Samuel West when he speaks contemptuously of it as follows: "Why this theory is mentioned in every book it is hard to understand, for at the most it would account for the change in shape only, and this is neither constant nor essential."

The theory is based on the alteration of form which the thorax undergoes in typical examples of emphysema. Normally the chest is slightly flattened in its antero-posterior diameter. The ribs are united to the costal cartilages in such manner that a line drawn through the longitudinal axis of the ribs and cartilages forms an angle which, very obtuse at the summit of the chest, becomes more acute as the ribs descend toward its base (Fig. 38). When inspiration is made and the thorax expands the ribs rotate somewhat on their long axis, the elastic costal cartilages elongate, and at the height of inspiration form a still more obtuse angle with their respective ribs.

The antero-posterior diameter of the chest is thus increased and its contour is more nearly round. This is, in fact, the shape which the emphysematous thorax displays and to which is applied the term barrel-shaped chest.

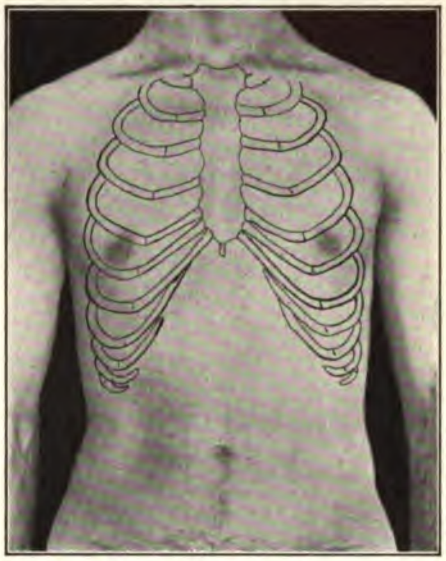


FIG. 38.—Normal relation of ribs and sternum.

Freund concludes that this change of form is due not to a primary expansion of the lungs to which the thoracic walls have accommodated themselves, but is the result of structural changes in the costal cartilages. To quote from Hertz, "Somewhat short of the twentieth year, according to Freund, the ribs assume, from their central axis outward, a dirty yellow color, and become softened, stratified, and coarse-fibered. As these fibers now have a tendency to bend and twist, roundish spaces and breaches form between them. The cartilage is thus pressed apart in its outer layers, and increases in volume in all directions. The tissue loses its flexibility and elasticity, becomes stiff and brittle. On account of this wayward growth, the two bony points, one upon the rib and the other upon the sternum, between which the cartilage is inserted, are spread further apart, and thus the ribs are forced outward and upward, and the sternum forward and somewhat downward. By the further increase, the cartilage undergoes an exaggeration of its natural outward convexity, and accommodates itself to the level of the anterior extremities of the bony ribs, so that the thorax, as regards the six lower true ribs, loses its normal expiratory attitude, and assumes permanently that of inspiration. The permanent distention and enlargement of the thorax thus produced may lead to secondary distention of the lungs, and finally to emphysema."

The only other theory requiring mention is that of Lange who attributes emphysema to a neuromuscular paralysis of the bronchioles. Even if such a paralysis were capable of producing dilatation of the alveoli, it is impossible of satisfactory demonstration as actually occurring, and has not so much to recommend it as have the other theories to which more space has been given.

Morbid Anatomy.—The chest being opened the lungs fail to contract but are observed to stand forth with unusual prominence. Their anterior borders are contiguous throughout, covering over and concealing the pericardium from view. The full rounded apices extend high above the clavicles while the

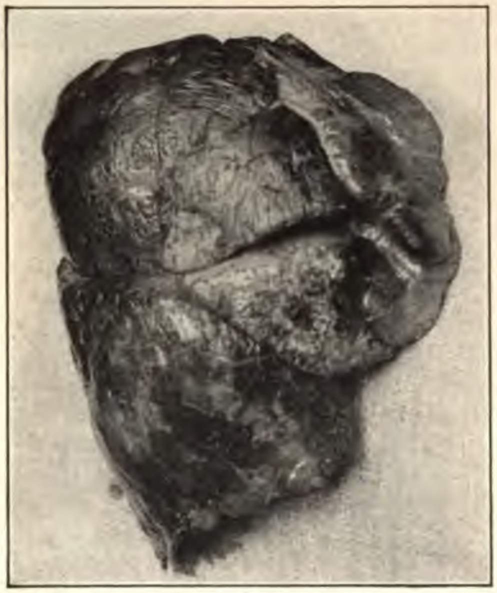


FIG. 39.—Lung of pulmonary emphysema.

ferior margins reach unusually low, in well-marked instances to the level of the eighth or even the ninth costal cartilages.

The lungs look much dryer and paler than natural, feel abnormally soft and downy, and do not crepitate as they should on being squeezed between the fingers. Scattered along their margins are elevations which have the appearance of blebs and which, on closer examination, are seen to be subpleural sacs or dilatations formed by extravasation of air into the interstitial connective tissue. These collections of air form what is known as interstitial emphysema, in contradistinction to the substantive variety now considered.

Upon the removal of the lungs from the thorax they are seen to be in general more voluminous than usual (Figs. 39 and 40) and often to present on their surfaces transverse ridges and depressions corresponding to the position of the intercostal spaces and ribs, and caused by the distention and consequent strong pressure against the chest-wall. The lungs may or may not have been united to the parietal pleura by old adhesions. Areas of interstitial emphysema are often found at the roots of the lungs as well as at their margins. These blisterlike formations vary in size from that of a pea or a cherry to that of a lemon or a small orange.

Upon section the cut surface is seen to be paler and more bloodless than normal, and here and there minute

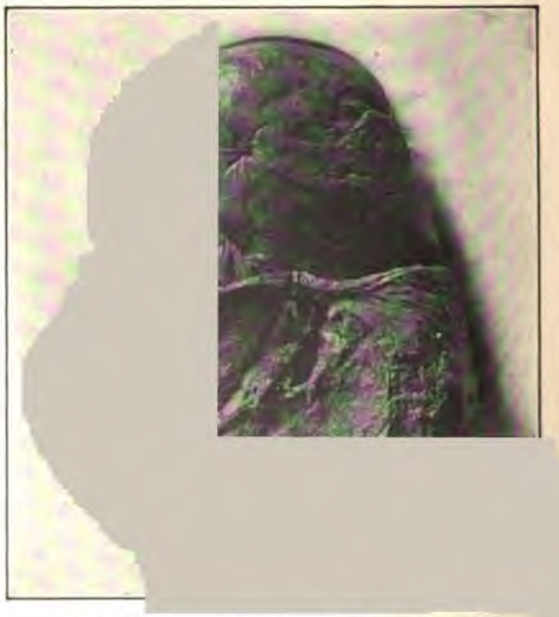


FIG. 40.—Lung of pulmonary emphysema.

inspection may detect tiny cavities which give to the organ a somewhat spongy appearance. The mucous membrane of the bronchi generally presents more or less evidence of chronic inflammation, being thickened, congested or thrown into longitudinal ridges, and covered here and there with masses of tenacious yellow secretions. Bronchiectases are not generally discovered in this diffuse variety of emphysema.

Examined microscopically (Fig. 41) the alveoli and infundibula are seen to be dilated and their walls thinned. Here and there, indeed, the atrophy of the sæpta has gone so far as to cause perforation and coalescence of adjacent alveoli, i. e., the minute cavities already mentioned. It is also apparent that the blood-vessels in the alveolar framework have become attenuated or have even disappeared, which accounts for the dry bloodless appearance of the emphysematous lung. Thickening of the interalveolar connective tissue is not present in typical cases.

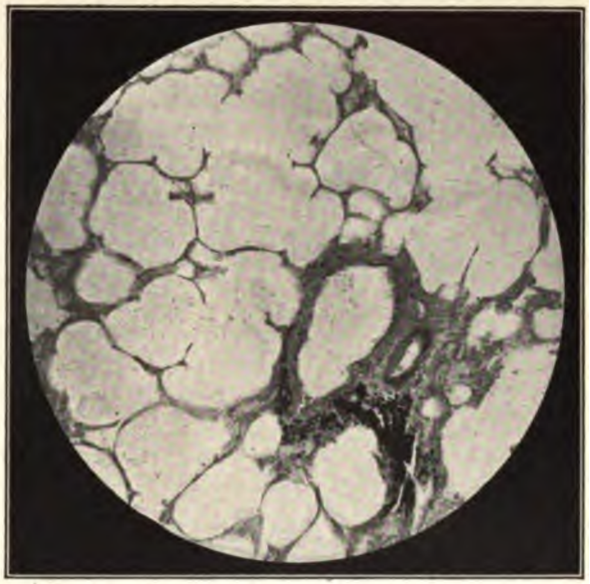


FIG. 41.—Microscopic section of emphysematous lung. Note anthracosis.

The associated changes are hypertrophy and dilatation of the heart, in particular of the right ventricle, possibly other cardiac lesions, and more or less sclerosis of the pulmonary arteries.

Symptoms.—Dyspnœa is the chief symptom from which emphysematous patients suffer. In minor degrees of the malady this is a breathlessness which declares itself only when some unwonted exertion is made, as hurrying across a street, mounting stairs or rising quickly from a chair to leave the room; but after the emphysema has become pronounced there is more or less shortness of breath even in repose. When, at length, the disease has reached an extreme degree, the air-hunger manifests itself as urgent dyspnœa, shown not by labored respiratory movements, which indeed are rendered impossible by the permanent distention of the thorax (Fig. 42) and by the lowered position of the diaphragm, but by the prominence and tugging of the sterno-cleido-mastoid and other cervical muscles, and by the cyanotic, anxious appearance of the face.

This dyspnœa, so inseparable from emphysema, is partly of mechanical partly of chemical origin. The unnatural increase in the diameters of chest which gives it the barrel-shape of extreme inspiration renders fur expansion impossible and prevents the in- and out-rush of air, so that the proper exchange of gases within the lungs does not take place. Carbonic acid accumulates in the blood, and through its impression on the respiratory centers creates a feeling of air-hunger. In the earlier stages of the disease sufficient oxygenation of the blood takes place during repose, and it is only during periods of exertion, when the demand for oxygen is greater than the supply, that breathlessness is experienced. When however the destruction of pulmonary capillaries and the rigidity of the chest-walls have restricted the flow of sufficient blood to the lungs, hæmatosis is deficient at all times and dyspnœa is constant. In this stage, expiratory collapse of the lungs is so impeded that patients cannot even blow the nose or cough efficiently.

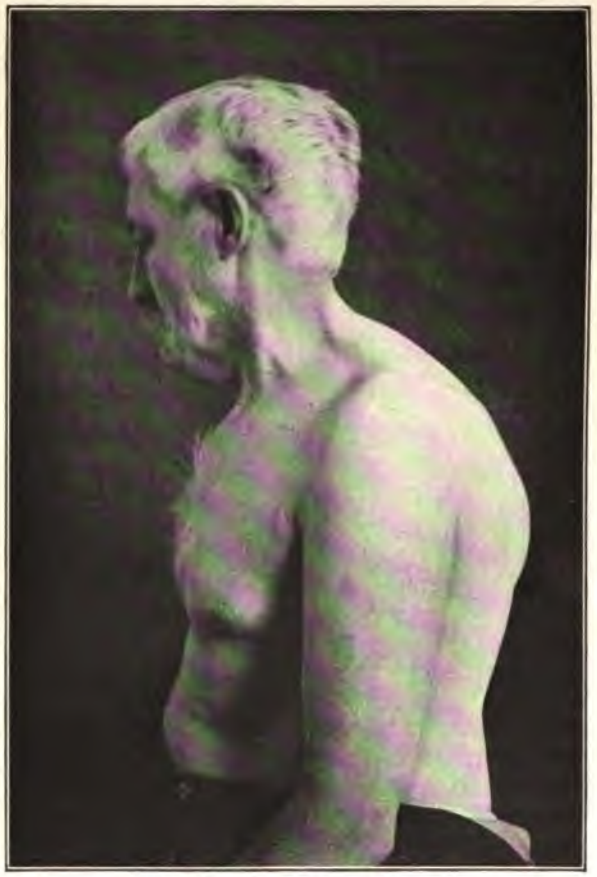


FIG. 42.—Chest of emphysema.

Circulatory embarrassment is now added to respiratory inadequacy and signs of venous stasis grow pronounced. The countenance becomes puffy and cyanotic, distended capillaries appear on the cheeks and nose, the external jugulars stand forth distinctly and may even show pulsation. The pulse is small, feeble and accelerated, and examination of the heart reveals dilatation of the right chambers.

The heart-tones are feeble, often obscured by a soft blowing tricuspid murmur, or perhaps over the right ventricle they possess a galop rhythm. The liver is swollen and palpable, usually painful and tender, particularly in the median line. The urine is scanty and often albuminous, hæmorrhoids develop and œdema appears at the ankles.

The sufferer has been compelled to take to his bed where, supported in a sitting position, he presents a clinical picture of dire distress tormented by orthopnœa and a desire to cough which he cannot suppress and yet cannot accomplish successfully for want of expiratory power. Indeed, the chronic bronchitis which, in most cases, becomes added to the emphysema, forms a very prominent and sometimes early feature, plaguing the patient and augmenting his difficulty of breathing by the accumulation of mucus in the finer tubes and by increasing the emphysematous distention of the lungs.

Years before the termination of their disease, patients are incapacitated for work and hence, in the poorer classes, drift into hospitals and almshouses where, sheltered from the vicissitudes of weather and recipients of medical aid, they remain often for years.

I have already mentioned a man who had been a sailor and who, so long as he could remain in one of our city hospitals, dragged out an existence of tolerable comfort except when, during the winter, his bronchial catarrh became aggravated. Then, for a time, his suffering grew intense and he was confined to his bed with frequent ineffectual cough and distressing shortness of breath. He would rally, at length, and again get about and even be able to sit out of doors on warm sunny days.

The COURSE OF THE DISEASE is essentially chronic and may drag through a number of years, each returning season finding the sufferer a little more feeble and more dyspnœic. Acute intercurrent affections, as bronchitis but in particular pneumonia, are very common causes of death; but when not carried off by such complications the victims of emphysema generally succumb to slowly increasing, general or cardiac asthenia, dying under the appearance of slowly advancing cardiac disease, with intense, often paroxysmal dyspnœa, cough and dropsy.

The following case is a fair example of the disease as it is encountered in private practice in the United States. Mr. M., a resident of Georgia, sought my opinion in June, 1903, because of his shortness of breath which threatened to incapacitate him entirely for the practice of his profession of law. He gave his age as sixty-five and stated that he had served as an officer in the Civil War, at which time he had been wounded. Up to about ten years ago he had enjoyed robust health and been a very active, energetic man.

He then developed a cough which had gradually grown more troublesome and which, of recent years, had been accompanied by distressing breathlessness. He had been a heavy smoker and a moderate user of whisky until he had a severe illness in February, 1903, since which time both habits had been abandoned. The illness referred to was one of cough, fever and great prostration which kept him in bed for several weeks. He thought it might have been pneumonia. Since then he had felt much weaker than before, and his breath was still shorter. Family history was negative and there was no further anamnesis worthy of note. Venereal disease was denied.

As soon as this patient entered my office and seated himself I perceived he was breathing with considerable difficulty. His respirations were audible several feet away and inspiration was accompanied by a slight wheeze. He spoke with apparent effort and his voice had a husky quality. After he h
remained quiet for a few minutes his dyspnœa disappeared, as far as co

be observed, yet at once became manifest upon his rising to undress for an examination.

The countenance was suffused, looked slightly puffed, and capillaries showed plainly on the cheeks and nose. The lips were somewhat blue. The neck looked rather full, and the chest had a distinct though not extreme rounded appearance. With each inspiration the chest was lifted slightly upward while the epigastrium sank visibly and evinced distinct pulsation directly below the ensiform.

The apex-beat could not be determined, but the pulse showed that the heart was beating ninety-two times a minute. The pulses were symmetrical, regular, with exception of three waves which followed each other in rapid succession, and were tense. The blood-pressure as registered by Gaertner's tonometer was 158 mm. In volume the pulse was rather small, and the arterial coats felt moderately stiff.

Percussion brought out a full resonant note which, on strong percussion, became unnaturally loud and drumlike. The præcordium was resonant, with absence of the area of superficial dullness, and the upper portion of the lungs behind yielded a far more resonant and intense note than is usual in these regions. The line of liver dullness in front was somewhat depressed, and excursion movements were very limited. The heart could be outlined with difficulty, but was shown to be increased in all diameters, especially to right and downward.

On auscultation the breath-sounds were rather diminished, now and then accompanied by a faint wheeze, while the expiratory portion was prolonged to about the length of the inspiratory. The cardiac tones were distant and clear, the aortic second being rather booming. Cough, which, by the way, was infrequent and feeble, brought out a few fine crackling râles at the posterior bases, but on the whole adventitious sounds were not pronounced.

Palpation of the abdomen revealed a thin dense hepatic border a hand's breadth below the costal margin, and pressure upward on the organ shut off the man's breath appreciably and called forth gentle remonstrance.

The urine was not examined for the reason that he had no nocturnal micturition, and he stated it had been found negative by his family physician a few weeks previously. Nevertheless, judging from the cardio-vascular changes it was inferred that there was some degree of interstitial nephritis.

The chief interest to me in this case of emphysema lay in its probable ætiology. There seemed to be nothing in his profession as attorney to produce it, and an inherent nutritional defect could also be excluded since it did not begin to show itself until after his fiftieth year. Likewise it did not seem reasonable to assume pulmonary stasis as its predisposing cause, since there had been no embarrassment of breathing, as far as could be ascertained, until after the development of his cough ten years before my examination.

On the whole, therefore, it was most in accord with the history to assume an initial bronchitis to which, in time, the emphysema gradually became secondary. This hypothesis appeared strengthened by the absence of signs of profuse bronchial catarrh, and, it will be remembered, Hoffmann maintains that emphysema is most apt to develop in this very class of cases, namely, such as have bronchitis of the finer tubes with scanty difficult expectoration.

Finally, one other fact worthy of comment was the almost complete absence of any other symptom than shortness of breath. This was not at all asthmatic, i. e., paroxysmal, but was a veritable breathlessness on even slight exertion. In repose he was comfortable, but as soon as he stood up or engaged in forensic argument his dyspnœa asserted itself. The action of the heart showed that this was not the organ at fault, and hence the difficulty of respiration could not be attributed to anything else than the mechanical hindrance occasioned by the distention and immobility of the lungs.

In completing the clinical picture of diffuse emphysema it should be stated that the symptoms often become changed by attacks of asthma. When such is the case it is my observation that the paroxysms lack periodicity, that instead the dyspnœa, never wholly absent, manifests irregularly recurring exacerbations of a veritable asthmatic type. When things have reached this pass the sufferer's condition is truly pitiable. In addition he is plagued by cough, is unable to take adequate nourishment or, if he does eat, is bloated with gas which causes frequent eructations and increases his dyspnœa. He loses strength and at length is confined to the house or bed in a state of suffering which makes death welcome. Such cases drag along for years, but may at any time be terminated by acute bronchitis or pneumonia.

Physical Signs. **Inspection.**—In well-marked cases the chest assumes a characteristic contour to which has been given the name of the "barrel-shaped chest." The sternum and attached costal cartilages, particularly below, are thrown forward so as greatly to increase the antero-posterior diameter of the thorax and give it a rounded appearance. The spinal column is lengthened and curved strongly forward, so that the scapulæ are spread apart and the patient looks round-shouldered (Fig. 42). His face is puffy and congested, the neck sinks into the shoulders, the superficial veins are engorged, and usually there is visible pulsation in the pit of the stomach.

If the patient's breathing is watched, it is noted that although the cervical and other auxiliary muscles of respiration contract strongly the thorax does not expand and collapse with each act, but is lifted upward while the epigastrium sinks in during inspiration, to protrude again with expiration. If dyspnœa is extreme there may even be coincident depression of the cervical regions, the same as in asthma, while a horizontal groove may sometimes be seen at the level of the lowest ribs. In a word, the chest appears to be in the position of permanent inspiration.

Palpation.—Vocal fremitus is diminished or sometimes abolished, and the impulse of the heart is indistinguishable in its normal situation. Instead there is usually a distinct epigastric pulsation.

Percussion.—This affords the only reliable sign of vesicular emphysema. Owing to the distention of the alveoli the lungs are found highly resonant throughout, being drumlike or hyperresonant, even in parts that normally possess some degree of impairment. The area of absolute cardiac dullness is obliterated and the resonance extends far below its normal limits. It may, indeed, reach even to the inferior costal margin, replacing both hepatic and splenic dullness.

It is not, however, upon exaggerated pulmonary resonance that the diagnosis is to be based, for such may sometimes be observed in persons with voluminous lungs and thin thoracic walls, or in those who have resided at a high altitude. The *pathognomonic sign is the lowered and stationary position of the diaphragm.* To determine this, one notes the situation of the superior boundary of liver-dullness during quiet respiration and then, getting the patient to take a deep breath, percusses the base of the chest to perceive to what distance the hepatic flatness has descended. Then, as expiration occurs, it is again determined by percussion how far this line rises. The difference thus shown constitutes what is known as the excursion-movement of the diaphragm. This differs greatly in different individuals, but in health is always very appreciable, deep inspiration forcing down the line of hepatic dullness at least to the upper border of the seventh and often the eighth rib in the mammillary line. In vesicular emphysema, on the contrary, this alternate descent and ascent of the diaphragm does not take place, and if the hyperresonant lung reaches constantly too low, as well as remains stationary, this fact is diagnostic of pulmonary emphysema.

Old pleuritic adhesions may hold the lung-border fixed, but in such a case the upper line of hepatic dullness is apt to be permanently raised instead of lowered, and inspection shows an absence of Litten's shadow sign (see Chronic Pleurisy).

Auscultation.—The breath-sounds are vesicular and usually of diminished intensity. Expiration is lengthened, being in some instances quite as long as is inspiration. If râles are present they are due to associated bronchitis, and for the most part are wheezing. The heart-sounds are feeble because obscured by the overlapping lung. If dilatation of the right ventricle has been produced there is a systolic whiff in the tricuspid area. Organic lesions of the heart, if they coexist, will be declared by the auscultatory evidence of such lesions.

Diagnosis.—In well-marked cases of diffuse vesicular emphysema diagnosis is not difficult, but cases are not infrequently encountered in which it is not so easy, and then attention must be paid to the following points: (*a*) History and evidence of shortness of breath especially on exertion, the dyspnœa being of gradual development and due to defective expiratory power, and generally associated with more or less cough. (*b*) In a child or youthful person, the history of previous acute disease as pertussis or measles; in an adult, history of some occupational influence or of antecedent chronic bronchitis, possibly also of similar affection in other members of the same family. (*c*) The barrel-shaped chest and other signs described above under inspection. (*d*) Resonance on percussion usually exaggerated, replacing præcordial dullness, depressing the line of hepatic flatness and causing marked restriction or complete abolishment of the normal excursion movement of the diaphragm. (*e*) Feebleness of breath-sounds with lengthening of the expiratory portion and usually with bronchitic râles.

Two things are to be especially borne in mind, namely, the barrel shape of the chest is not invariably or necessarily present, and, on the other hand, when present may be the result of previous hard labor without any emphysema. It is of significance only when associated with symptoms and percussion signs of vesicular dilatation.

One should not rest content with having made a diagnosis of this condition. He should also endeavor to ascertain whether or not some other pulmonary disease may not be masked under the guise of emphysema. This is especially true of pulmonary tuberculosis since this is not infrequently associated. If such be the case, there ought to be evidence of the tuberculous affection in dullness, in a sense of increased resistance and fine crackling râles at one or both apices, and in bacilli contained in the sputa. There is also likely to be some increase of body-temperature in the latter part of the day, whereas in emphysema alone fever is absent.

An old abscess cavity may likewise be concealed by extensive compensatory emphysema, and may cause vesicular dilatation, though less marked, of the opposite lung. I have such a case in mind in which pneumonia is known to have terminated in abscess thirty years ago, and in which the signs of vomica are completely obscured by those of well-marked vicarious emphysema. The other half also of the thorax yields a hyperresonant note, so that without knowing the history, the case might very easily pass for one of substantive emphysema, particularly as the individual displays dyspnœa of effort from an enfeebled heart, and has a chronic cough from catarrh of the nasopharynx.

Differential Diagnosis.—Since the disease in question is characterized mainly by shortness of breath on exertion, it has to be distinguished from all other affections displaying the same symptom. It seems to me, therefore, that in this respect it is most likely to be confounded with primary cardiac incompetence from whatever cause. Error in diagnosis may arise, however, in connection with other diseases causing dyspnœa. Furthermore, since some of the signs are similar, difficulty of differentiation may arise in cases of pneumothorax. Emphysema and chronic bronchitis may be and often are so intimately associated as to render it extremely difficult, if not actually impossible, to determine which of the two is primary or more important. These various affections will be now considered *seriatim*.

(1) *Cardiac Dyspnœa.*—(*a*) History. Careful inquiry may elicit ætiological or other facts pointing to the probable existence of cardiac disease prior to the development of dyspnœa. (*b*) Symptoms. The breathlessness on exertion is very like that of emphysema; but when dyspnœa of such degree is present, there are likely to be also other evidences of more profound circulatory disturbance than in most instances of primary emphysema. (*c*) Physical Signs. These are more pronounced on the part of the heart than of the lungs, namely, murmurs and signs of cardiac dilatation with increase of absolute præcordial dullness and rigidity of the thorax, hypostatic pulmonary congestion, rather than hyperresonance or obliteration of cardiac dullness, and other evidence of mechanical distention of the lungs. If emphysema and secondary cardiac dilatation with inadequacy coexist, it may be impossible to assert which of the two conditions is primary.

(2) *Aortic Aneurysm.*—(*a*) History. Syphilis, strain, age, and other factors pointing to the probability of aneurysm. (*b*) Symptoms. Pain, inten

paroxysmal cough, dyspnœa, often spasmodic and influenced by posture as well as or, perhaps, more than by exertion, and the difficulty of breathing inspiratory rather than expiratory. (*c*) Physical Signs. Pressure on lungs and blood-vessels, and interference with the proper expansion of one or of both lungs, rather than signs of overexpansion. (*d*) The X-ray may reveal the presence of an intrathoracic tumor.

(3) *Pneumothorax.*—(*a*) History. That of previous wasting, fever, sputa and other evidence of tuberculosis. (*b*) Symptoms. Sudden pain in the side rapidly followed by difficulty of respiration, shock, etc. (*c*) Physical Signs. Unilateral distention with tympanitic resonance, amphoric breathing on same side, displacement of heart to opposite side and of liver or spleen downward. If fluid be also present, the characteristic splashing sound and the bell or coin sound above the level of the fluid.

(4) *Chronic Bronchitis or Emphysema the Leading Condition.*—As previously remarked this is often a very difficult question to answer as well as an important one from the standpoint of treatment. The following point may be of aid. (*a*) History. That of Winter cough with relatively copious expectoration for years prior to the occurrence of much dyspnœa, and also history of occupation exposing to inhalation of dust rather than to increased expiratory effort. (*b*) Symptoms. Complaint of cough and mucous or mucopurulent expectoration rather than of breathlessness, although this latter may also be present. (*c*) Physical Signs. Pronounced bronchitic râles of all sorts, often over the larger tubes and predominating over the signs of pulmonary distention.

Lastly it should be remembered that emphysema and asthma often coexist, for the twofold reason that old sufferers from true bronchial asthma are very apt to become emphysematous, while, on the other hand, victims of emphysema and bronchitis often develop asthmatic attacks. In all such cases it is important to determine which is the primary affection, and it may be by no means an easy task. The differentiation must depend chiefly upon the history. If the asthma has existed from early life, it is clear that this is primary, but if it has come on late in life, it may have followed the emphysema. Even then much must depend upon the history, and one should take great pains to ascertain if, perchance, there were not asthmatic paroxysms with intervals of comparative immunity for years or months before the patient began to notice habitual dyspnœa of effort with spasmodic exacerbations of dyspnœa. In such an event it is, of course, presumable that the emphysema was secondary. If it was primary, then, of course, the breathlessness and cough preceded the onset of asthmatic seizures.

Prognosis.—Diffuse vesicular emphysema is essentially a chronic disease. Ordinarily it is slow in its development and slow in its progress, running over a period of years. In its milder manifestations it does not threaten life directly and it may not seriously incapacitate the individual for the performance of light occupations. It is progressive, however, and unless it can be checked by treatment will, in time, lead to invalidism.

When the disease is pronounced it is not likely to be materially improved by treatment, and in this respect furnishes an unfavorable prognosis. In the majority of cases an amelioration of symptoms is the most that can be held out to the patient. The association with chronic bronchitis renders prognosis still more unfavorable, since each condition acts to the detriment of the other. If the emphysema is attended by or has led to serious cardiac incompetence, there is practically no prospect of more than temporary relief by the most skillful management, and a fatal issue is only a question of time, perhaps of only months or weeks.

The supervention of asthmatic paroxysms is a bad feature since, even if these do not shorten life, they are apt to resist treatment and serve to aggravate the emphysema. Emphysematous patients are liable to attacks of acute bronchitis but not pneumonia (Liebermeister). Since the powers of resistance are already taxed by the primary disorder, such intercurrent diseases are apt to prove more than their feeble vital forces can withstand. All such complications should be regarded, therefore, with grave apprehension.

The prognosis may also be considered most unfavorable if the emphysema appear early in life. It is perhaps true that young children may outgrow it, but its development in early adult life probably indicates a degree of nutritive disturbance which will cause the disease to progress more rapidly than at middle or advanced age. In such, moreover, there is small likelihood of benefit from treatment of any sort.

In a word, although life may not be actually threatened by emphysema, except in its late stages, still there are so many conditions which may work against betterment that it is never wise to promise much from therapeutic management.

Hoffmann states that Waldenburg based the prognosis on the results of spirometric observations, it being very unfavorable when this apparatus showed the vital capacity reduced to one half or less. Practice has much to do with the use of this instrument, however, and judgment should be passed only after the patient has familiarized himself with its use.

Treatment.—The great aim of management in any affection is its cure. Unfortunately this is scarcely possible in diffuse emphysema, since it involves the detection and removal of the cause as well as the removal of pathological changes which are manifestly incurable. In the first place, it is not always possible to determine the precise ætiological factor, and in the second place, this cannot be removed in every case when discovered. Thus, an inherent nutritive defect is something which can only be inferred, or, if substantiated, cannot be removed. In such a case the most that can be done is to minimize or remove such other influences as are harmful in the presence of a nutritional weakness.

It is likewise not always possible to cure a chronic bronchial catarrh or to get a patient to abandon his injurious occupation. His subsistence and that of his family depend upon his employment and he may be unfitted for any other kind of work. Nevertheless, in a given case of developing emphysema an attempt in this direction should be made. Failing in the removal of the cause, the physician must next direct his efforts to lessening its evil effects.

Bronchitis must be treated in the hope of diminishing the injurious cough. The patient must be impressed with the necessity of restraining his cough, and when this is uncontrollable, of accomplishing the act in the most effective and at the same time least injurious manner. This is to be done by augmenting expiratory effort in the way described in the treatment of chronic bronchitis, namely, by compressing the thorax by the arms. Remedies of service in chronic bronchitis without emphysema are applicable also to the class of sufferers now considered.

In the management should also be included treatment by certain mechanical devices, i. e., the Pneumatic Differentiator and Waldenburg's compressed-air apparatus. The former is an iron cabinet so constructed that the patient sits inside and yet is supplied with air from outside the box. The door and window through which the tube for respiration passes are capable of being hermetically sealed, while by means of a bellows on the top of the cabinet the pressure of the atmosphere within can be raised or lowered at the will of the operator. The Waldenburg machine is simply an apparatus for the inhalation of compressed or rarefied air.

If, in the treatment of emphysema, the Differentiator is used the following plan is adopted, based on the indication for augmenting expiratory power which, owing to distention and rigidity of the thorax, is lessened or nearly lost. The patient enters the cabinet and takes his seat, facing the window in front. The door is now tightly closed and the patient, placing between his lips the tube which passes through the window, begins to breathe. The operator with his hand on the arm of the bellows watches his respirations and each time an expiration is made suddenly shuts the bellows and thus compresses the air around the patient's body. Inasmuch as the air he respires is at ordinary atmospheric pressure and that surrounding his chest is raised to perhaps two atmospheres, the difference is considerable, so considerable, in fact, as to prove a very powerful aid to expiration. Although some years ago I possessed such a Differentiator I had no experience in this treatment of emphysema. I have been assured by others, however, of its efficacy, and this mode of treatment certainly appeals to one as theoretically applicable to this class of sufferers. It is obviously of real service, however, only when the emphysema is in an early stage and atrophy of the alveolar sæpta has not taken place.

Another method of treatment which in Germany has been much employed and scientifically investigated is that by means of Waldenburg's apparatus and others of similar design. Instruments of this kind are constructed on a different principle from that of the Differentiator, since, instead of aiding expiration by compression of the chest, they only permit the inhalation of compressed air or exhalation into rarefied air. The former method is not applicable to cases of emphysema unless there be much associated bronchitis and alveolar dilatation is not extreme. Even then the inhaled air should be compressed only $\frac{1}{80}$ to $\frac{1}{40}$ of an atmosphere and exhalation is made into the surrounding atmosphere.

Waldenburg claimed remarkable results in emphysema by having patients expire into rarefied air, i. e., a partial vacuum, $\frac{1}{80}$ to $\frac{1}{60}$ of an atmosphere. Theoretically such a procedure ought to tend to empty the lungs of their residual air or such a portion of it as would make the succeeding inspiration all the fuller. As a matter of fact, however, it would seem from the investigations of others (Josephson, Lazarus, Schreiber) that instead of the vital capacity being increased by such a procedure it is actually decreased. Schreiber found that although it was possible for a healthy person to exhale 150 c.c. more of air into a partial vacuum than into the outside atmosphere, still there was no appreciable diminution in the size of the chest.

Schreiber concluded that benefit from such a method of treatment can only follow in those cases in which there is an acute or a subacute dilatation of the air vesicles. He believed, furthermore, that in cases of pronounced and long-standing emphysema actual harm might ensue from the suddenly augmented flow of blood within the pulmonary capillaries incident to the diminution of air-pressure within the alveoli and to the intensification of respiratory movements. On the whole, therefore, this, as well as the treatment by means of the Differentiator, is suitable only to early cases and promises nothing more than possible injury when there is probably atrophy of the alveolar sæpta.

The possession of either of the mechanical devices just described is not always possible to the general practitioner, since they are expensive and occasion for their use does not often arise. In lieu of such appliances it is recommended that attempts be made to assist the feeble expiratory power of emphysematous patients by systematic compression of the thorax after the manner suggested in a preceding paragraph.

An assistant may powerfully compress the chest simultaneously with the patient's expiratory act or the latter may be instructed how to do this himself, namely, by bringing his arms down across the front of his chest while at the same instant he bends forward and thus aids expiration by compression of his diaphragm. Such measures are manifestly useless and may be positively harmful in cases of substantive emphysema in which the chronicity and severity of symptoms are such as to suggest the probability of consecutive atrophy and rupture of alveolar walls.

It is likely that when serious pathological changes are present, too vigorous attempts at compression of the thorax may damage the dilated lungs still further. It is not merely theoretical, moreover, to assert that such attempts would seriously embarrass the already overburdened right heart and aggravate existing venous stasis. These considerations make it plain that mechanical treatment of emphysema should be confined to the early stage of the disease. They would be of special service in young children whose tissues are still elastic and capable of restoration to their previous healthy state.

Along the same line is a recommendation I recollect having seen many years ago by a French physician whose name, I regret to say, I cannot now find. This was, that emphysematous patients should wear continuously a steel band about their chest for the sake of the constant pressure on the thoracic parietes exerted by such a device. It is not impossible that habitual compression of this kind might, in time, diminish or at least prevent an increase in the distention of the chest which, if it cannot be said to cause, at all events accompanies many cases of emphysema. A heavy elastic bandage of rubber, while less powerful and durable than one of steel, might yet tend in the same direction. It would at least encourage the patient in the feeling that his physician was not merely a passive spectator of his physical condition.

The *hygienic management* is of the utmost importance, whether it be in the early stage, with a view to prevention of more serious manifestations, or in the extremer degrees of the disease. Injurious influences of all kinds must be removed, when possible, and when not possible must be minimized. If chronic bronchitis seems to be the harmful factor in the production or the aggravation of the emphysema, the patient must be withdrawn from all conditions, as dusty occupations, exposure to inclement weather, unsanitary surroundings, etc., which are known to keep up the bronchitis. The medical adviser must not content himself with merely prescribing medicine but, by minute inquiry, must inform himself concerning his patient's environment, and then must aid by advice and in other ways in getting him into surroundings calculated to improve his physical condition. For particulars on this head the reader is referred to the chapter on chronic bronchitis.

The *diet* should be included in the hygiene of the daily life and is a matter of considerable importance with emphysematous patients. It may not directly influence the disease and yet the necessity of adequate nourishment cannot be doubted in any chronic malady, especially one in which there may be an inherent weakness of tissue. The nutritional defect assumed to underlie emphysema does not depend primarily upon a lack of nutritive supply, and yet insufficient or improper food may very well accentuate such a weakness of tissue. Such must undoubtedly be the case in children, and hence in patients of tender years, convalescent from pertussis or measles; the dietary must be ample in quantity, nutritious in quality and easily digested. It should be especially rich in fats and phosphates, and hence milk, cream, eggs, cereals and meats are indicated. But no matter how excellent may be the dietary it cannot build up a weakly patient unless it be combined with an abundance of fresh air and sunshine. The patients must therefore be encouraged to spend as large a part of each day out of doors as possible, avoiding, of course, undue exposure in weather and seasons likely to induce or aggravate bronchial catarrh.

Climate as a factor in the management of emphysema must be considered with reference to associated conditions and their causative influence on the alveolar dilatation rather than with regard to any power it may possess in retarding or curing emphysema directly. Any climate which is suitable to outdoor life is good for the emphysematous, provided the altitude is not high. In short, any resort at low levels suitable to chronic pulmonary disorders will answer for this class of patients. Preference should be given to localities which are bracing rather than enervating and are moderately dry, e. g., Algiers and the Riviera in Europe, and in the United States the piney regions of South Carolina, Georgia and Central Florida, Southern Texas, Phœnix, iz., in the Winter, and Southern California. Elevations of over 2,000 feet d seem to me inadmissible because likely to increase respiratory embar-

Many persons of this class may not, indeed, be able to endure evation, for the reason that their chests, already expanded to their utmost, are not capable of still further expansion in response to lessened atmospheric pressure. Low altitudes should be the rule, therefore, and yet the degree of emphysema, and hence of secondary dyspnœa, in any given case must determine this question.

Medicinal agents are not curative in this disease, and therefore are of service only as general tonics or for the relief of symptoms. Nux vomica and strychnine are universally recommended as the best tonic for the respiratory organs and may be administered for a long period, either alone or in conjunction with any other remedies that appear indicated. Ammonia and digitalis may be given when cardiac weakness becomes added to the emphysema. It should be remembered that strychnine or nux vomica must not be administered with ammonia preparations since, in alkaline solutions, the alkaloid is apt to be precipitated, when there is danger of the patient's getting an overdose as he nears the bottom of the bottle. It is a good plan, therefore, to add a little acid to solutions containing nux vomica.

Other tonics as iron and arsenic are also useful, especially the latter because of its well-known tonic properties in pulmonary affections characterized by dyspnœa. Cod-liver oil is frequently prescribed for emphysematous patients but is said not to be so well borne by those who are corpulent as by persons who are emaciated.

Symptoms calling for special treatment are generally such as arise from the associated bronchitis or from circulatory embarrassment. The former disease has been fully dealt with elsewhere, and hence it needs only to be stated here that the same measures are applicable when it is combined with emphysema as when the bronchial catarrh exists alone. Cough, when especially troublesome, should be mitigated by all means and to this end heroin seems particularly suitable. (See what is said on this subject in the treatment of chronic bronchitis.) When dyspnœa becomes very annoying this same remedy or even morphine may be given, since the indications for its use outweigh its drawbacks.

Digestive disorders with their flatulent distention of the stomach and intestines should receive careful attention by appropriate dietary and by remedies calculated to promote better digestion and to allay the formation of flatus. Constipation must not be permitted and an occasional cathartic often affords striking relief.

When cardiac asthenia is pronounced the same measures are indicated (rest, cardiac tonics and cathartics) as in cases of primary heart disease. The sufferers should be kept in bed and told not to engage in talking. If nocturnal dyspnœa sets in, nothing is so efficient as a hypodermic of a small dose ($\frac{1}{8}$ of a grain) of morphine with atropine.

CHAPTER XXIII

ATELECTASIS

By atelectasis or pulmonary collapse is designated a condition in which portions of one or both lungs or even the whole of one lung are devoid of air and the walls of the affected alveoli and even of the terminal air-tubes fall together or collapse. Prior to the contribution by E. Joerg (1832) this condition was regarded as inflammatory and was often confounded with pneumonia, but, thanks to Joerg, Traube and many others, it is now known to be purely mechanical and independent of inflammation, except in so far as this latter may be secondary to atelectasis.

The affection is usually divided into the *congenital* and the *acquired*, the former being but a persistence of the unexpanded condition presented by the lungs during fœtal life. This form does not differ essentially, however, from the acquired and hence the two will here be considered together. This is true of their ætiology as well as of their pathological anatomy, except that in the causation of the acquired form a far greater number of factors comes into play. Moreover, as pointed out by Hoffmann a newly born infant may breathe satisfactorily for a few hours and then, in consequence of weakness, cease to respire vigorously and hence allow its lungs to go back into the state of fœtal collapse; in which event there is nothing in the state of the collapsed lungs to distinguish them from lungs that have never been expanded.

Ætiology.—A great variety of conditions may serve to bring about pulmonary collapse, but when these are analyzed they are found capable of classification according to their *modus operandi*, as is done by Fraenkel. In the first group may be brought those conditions which act through deficient inspiratory force; in the second, those acting through obstruction of the air-tubes; in the third, such forces as act by compression. In some cases several such agencies may be operative.

In the strictly *congenital form* and in the atelectasis of the newborn, the collapse may persist or reassert itself in consequence of feeble inspiratory force, and is very likely to be the case in protracted labors or those requiring the use of forceps. In the latter it is very possible for the head, and hence the respiratory centers, to be injured. Feebleness of respiration is apt to be operative in weak infants and those born prematurely.

Another ætiological factor in newly born infants is the obstruction of air-tubes by vaginal secretions or meconium, and hence atelectasis from this cause is more likely in protracted labors. Finally, both of the factors just enumerated may be operative in the same case.

The *acquired form,* as a result of feeble inspiratory efforts, is met with most commonly in adynamic states, as rachitis, typhoid and other continued fevers. In such, the factor just mentioned is intensified by the protracted decubitus maintained by the patients, and hence the atelectasis is apt to be succeeded by the evidence of hypostatic congestion.

Pulmonary collapse from *obstruction* occurs in a variety of conditions, most obviously, of course, from foreign bodies in a bronchus. The occlusion of the tube must be complete, else emphysema and not atelectasis is likely to occur. This group of cases is seen most often in infants and young children suffering from capillary bronchitis and broncho-pneumonia, especially when allowed to lie too long in one position, but it may be met with, equally well, in adults enfeebled from chronic catarrh of the finer bronchial tubes. That it does not occur more often in chronic bronchitis is probably owing to the vigorous respiratory efforts and fits of coughing which serve to expel secretions that may be blocking the bronchioles. It is obstruction that is operative when atelectasis takes place in measles and pertussis, diseases serving as frequent ætiological factors in childhood. In these there is probably tumefaction of the bronchial mucosa as well as accumulation of secretions.

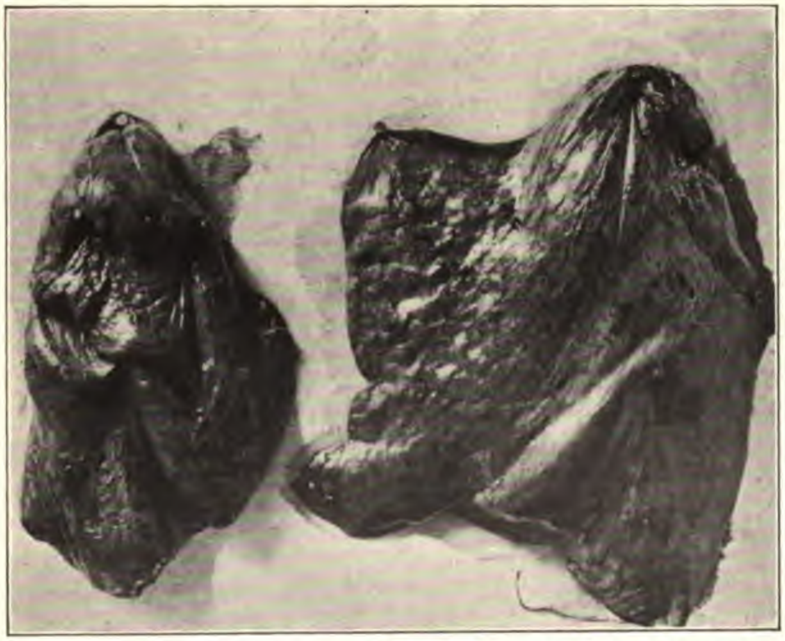


Fig. 43.—Atelectasis of right lung in unilateral hydrothorax. Note difference in size.

Causes of the third class are those acting through *compression of the lungs*: accumulations of fluid or air within the pleural cavity (Fig. 43), pericardial effusion, tumors of various kinds; thoracic aneurysm, malignant new growths of mediastinum, pleura or lung, enlarged mediastinal and bronchial lymph glands, etc. Even an hypertrophied thyroid gland situated within or extending into the cavity of the chest beneath the sternum is capable of exerting sufficient compression to bring about collapse of the contiguous lung borders, and in Chapter VIII is the reference to such a struma which powerfully compressed the trachea as well as the adjacent lungs.

In children, in particular, whose thoracic cavity is relatively small a greatly distended pericardium is a frequent cause of atelectasis, especially of the left lung, and the so-called Pins Sign is founded on the fact of collapse of the left lower lobe in extensive pericardial effusions. Fraenkel and others call attention to the fact that ascites, meteorism and intra-abdominal tumors are capable of producing more or less collapse of the inferior portions of the lungs through upward pressure and interference with the proper play of the diaphragm and other inspiratory muscles. Lastly, mention must be made of kyphoscoliosis as a condition capable of exerting sufficient pulmonary compression to bring about atelectasis, especially so, when in addition to the deformity, there is feeble respiratory power.

The causation of atelectasis would not be complete however if there were not still another factor at work, and this is absorption of the residual air by the blood circulating through the collapsed area. Proof of this lies in the observation that after death the lungs are not found atelectatic although retracted so far as is permitted by their elasticity. Virchow is said to have first suggested the absorption of the air as an indispensable condition, although it was Lichtheim who proved the correctness of Virchow's supposition by experiment. He also pointed out that it is this absorption of the residual air which completes the collapse produced in part by pleuritic exudates which are not so massive as fully to compress the lung. It is stated that first the oxygen, next the carbonic acid, and lastly the nitrogen are absorbed.

Morbid Anatomy.—Atelectatic areas may vary in size, from a small group of lobules to an entire lobe or even a whole lung (Fig. 44). Such areas are most frequent in the lower lung margins and throughout the lower lobes. They attract attention by their bluish color (in the congenital form brownish or grayish tint—Fraenkel), by the depression of their surface below the niveau of the surrounding lung, and by their firm consistence. Their cut section is smooth and when the blood-vessels have not shared in the collapse sufficiently to be largely emptied of their contents, blood may flow therefrom.

Atelectatic areas do not crepitate and they sink in water, yet they may be reinflated and become indistinguishable from the surrounding parts, provided further changes have not taken place. These changes are either the result of subsequent inflammation, i. e., broncho-pneumonia, due to the act of invading organisms, or develop in consequence of long-continued disu and consist in the development of interstitial connective tissue and compl desquamation of the alveolar walls. Circumscribed atelectatic zones are u ally surrounded and may even be obscured by areas of compensatory e. physema. Associated with the collapse are the changes of the primary ætiological condition.

Symptoms.—Except in the congenital form, pulmonary collapse is rarely if ever encountered or recognizable as an independent clinical entity. Consequently, such symptoms as it may occasion are likely to be masked by those of the disease on which it depends. Infants whose lungs are defectively expanded manifest lack of vitality, inability to cry loudly, and cyanosis. In young children suffering from broncho-pneumonia and widespread bron-

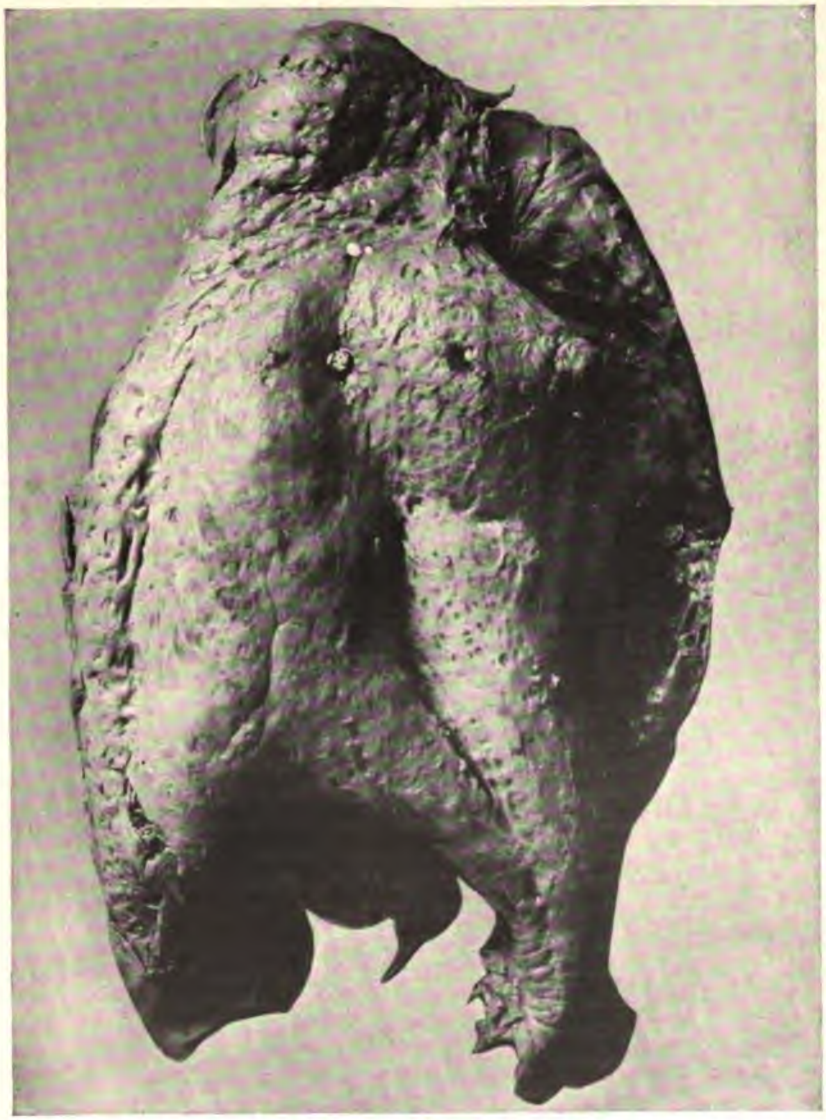


FIG. 44.—Right lung completely collapsed and adherent. But slightly reduced in size.

chitis, there is usually hurried and shallow breathing, but how much of this respiratory embarrassment is owing to the collapse and how much to the inflammatory disease is difficult of determination.

If, after subsidence of the active disease, the atelectatic areas do not reëxpand, the child is apt to remain feeble and evince more or less want of respiratory capacity. In affections in which bronchial obstruction or pulmonary compression is present, there is dyspnœa of an intensity proportionate to the extent of the atelectasis.

If the collapse is so persistent and extensive as to lead to hypertrophy and dilatation of the right ventricle, there is also more or less evidence of circulatory embarrassment shown by distention of superficial cervical veins and signs of right heart engorgement and visceral stasis.

Persons with pronounced kyphoscoliosis do not necessarily display symptoms due to pulmonary collapse or right ventricle strain. Indeed, they may, on the contrary, be capable of much physical exertion and enjoy good health. It is only when the heart shows signs of enfeeblement or when, after many years, the general health becomes impaired and respiratory energy suffers, that such individuals exhibit symptoms of pulmonary and cardiac embarrassment. Even then these are attributable to cardiac weakness rather than to the atelectasis as such.

Bedridden patients exhibit no subjective signs of the pulmonary collapse and the condition is, as a rule, first recognized by physical examination. The bases of the lungs exhibit more or less impairment of resonance, diminution of respiratory murmur and, on deep inspiration, fine crackling râles which disappear after the lobules have become inflated and resonance has returned. It may be stated, therefore, that the symptomatology of atelectasis consists rather in objective than in subjective manifestations.

Physical Signs.—Inspection perceives more or less rapidity and superficiality of respiration depending upon the extent of lung collapsed. In infants and children of tender age there is apt to be visible sinking of the soft parts during inspiration, especially at the base of the chest and, according to Leube, such retraction may be confined to the side corresponding to the lung involved. In cases of broncho-pneumonia or those in which the right heart suffers, there is ocular evidence of stasis and deficient hæmatosis, shown by distended veins and cyanosis.

Palpation.—This furnishes information of value especially in differentiating from pleuritic effusions, since atelectatic areas, if large enough to give rise to appreciable physical signs, favor an exaggeration rather than diminution of tactile fremitus. Palpation is of special value in infants in whom percussion may be very difficult and deceptive.

Percussion yields positive results only when the collapsed area is of sufficient extent (5 cm. in length according to Leube) to be recognizable by this means of examination. Such a zone yields dullness, but smaller areas are apt to be masked by a tympanitic note due to compensatory emphysema or relaxation of the lung. In young children, therefore, very light percussion is necessary to the recognition of such small dull patches.

Auscultation.—In children the collapsed lobules are apt to be concealed by the auscultatory phenomena incident to the primary affection, but when the atelectasis is recognizable, there are bronchial breathing, bronchophony and crepitant râles brought out by forced inspiration. With repeated forced inspiration these crepitant râles may sometimes be made to disappear and the respiratory murmur to become vesicular. Over extensive areas of collapse, breath-sounds are apt to be enfeebled and even abolished, as I have often observed in atelectasis due to compression by an aortic aneurysm.

Diagnosis.—The recognition of pulmonary collapse is usually not difficult. When, however, the diagnosis is not positive the existence of the condition may be inferred if some disease is present capable of its production. Given an ætiological factor, the diagnosis is rendered positive by the conjunction of dullness over a circumscribed zone at the base, feeble bronchial or diminished respiratory murmur, bronchophony, increased pectoral fremitus and crepitant râles, all of which give way to resonance, vesicular breathing and subsidence of râles after several deep breaths or change of position. In the case of aortic aneurysm mentioned above, a change in the direction of pressure led to partial return of pulmonary resonance and restoration of the breath-sounds which improvement in physical signs became still more marked when, after several months of treatment, diminution in the size of the aneurysm took place.

Differential Diagnosis.—In diagnosing atelectasis one must differentiate from the following affections:

(1) Small pleuritic exudates. This may usually be done by due attention to history and symptoms and to the evidence of pressure on adjacent organs as liver and heart. If change of posture causes a shifting of the area of dullness, and there is a lessening of pectoral fremitus as well as of breath- and voice-sounds, it speaks for pleuritic effusion and not collapse.

(2) Pneumonia of a limited area, and especially broncho-pneumonia, is to be distinguished by the history, the fever and the persistence of the signs, in spite of repeated forced inspirations, and by the character of the sputa. Since the physical signs of the two affections are very similar, especial attention must be given to the influence of repeated deep breathing on the dullness, bronchial respiration and crepitant râles.

(3) Hæmorrhagic infarct. In this the signs are also very like those of collapse, but when bloody sputum is ejected the diagnosis of infarct is clear. If blood is not expectorated, one must rely for differentiation on the permanence or disappearance of signs, as the case may be, after vigorous respiratory efforts.

(4) Tumors of lung or pleura. These are chronic diseases, unattended by fever. They give rise to symptoms which do not occur in pulmonary collapse, and by proper attention to these, as well as to the history, an accurate differential diagnosis ought to be achieved.

Prognosis.—This depends upon the cause of the collapse and upon its extent. The congenital form generally yields to appropriate treatment. In the young, when due to bronchitis, etc., it is likely to disappear with the return of the child to health, but if not and it is extensive, the general chitis, there is usually hurried and shallow breathing, but how much of this respiratory embarrassment is owing to the collapse and how much to the inflammatory disease is difficult of determination.

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CHAPTER XXIV

PULMONARY TUBERCULOSIS

Introductory.— Pulmonary consumption has probably existed as long as man has lived in established communities and ceased to lead a nomadic existence. At all events, its history is contemporaneous with that of medicine. The disease was familiar to Hippocrates and early medical writers who followed him, but, owing to ignorance of morbid anatomy and of proper diagnostic methods, was unavoidably confounded with other pulmonary complaints displaying similar symptoms. For centuries the ban of church and state placed upon *post-mortem* investigation restricted medical men to the clinical aspects of disease, and to this pulmonary consumption furnished no exception.

In the course of time, when anatomists and pathologists had broken away from the bondage to superstition and ecclesiastical prejudice, they began to study consumption from another standpoint. In 1689 Morton first ascribed the development of phthisis to masses in the lungs, which he called by the old term of tubercle. It is customary, therefore, to credit him with the discovery of tubercles as the cause of the various forms of pulmonary consumption. Such is, however, an error, since Morton did not recognize miliary tubercles but called by the name of tubercle the infiltrated and swollen bronchial lymph nodes seen in tuberculosis. Nevertheless, from Morton's observations as contained in his "Phthisiologia" dates the modern view of tuberculosis.

It was Bayle who, in 1810, first described miliary tubercles as we now know them, while his famous pupil, Laennec, emphasized the specific nature of pulmonary tuberculosis, denying that the lesions were of purely inflammatory origin. The half century following this great teacher was largely taken up with the discussion of the real nature of phthisis. Some believed the various lesions were all of the same origin, namely, tubercle; upheld by the weighty authority of Virchow, others, probably the majority, stoutly maintained the dual nature of pulmonary consumption, the tuberculous and the inflammatory form.

This was at a time when pathologists possessed no certain means of recognizing tubercle other than by its avascular structure and the giant cell first described by Langhans. Hence arose the extensive experimentation under the lead of Villemin for the purpose of determining whether or not animals could be rendered tuberculous by being inoculated in various ways with the sputa or other products of phthisical lungs. Although the results of such experiments were, for a time, conflicting it was at length satisfactorily demonstrated that Virchow's dual theory was untenable. Then, in the memorable year 1882, came Koch's announcement of his discovery of the tubercle bacillus. Thus a reliable means of identifying the true nature of pulmonary lesions was found, and the unity of pulmonary phthisis was proven.

Following the discovery of the tubercle bacillus medical investigation took a new turn. It seemed to therapeutists that now it would be possible to cure pulmonary tuberculosis by the introduction into the system of antiseptic substances which would antagonize or destroy the cause of the disease, and for the next few years medical literature was filled with all sorts of therapeutic measures. It was soon perceived, however, that medicinal treatment alone was inefficient and attempts were made to discover a remedy based on the principle of utilizing the antitoxins of the animal body.

About this time Koch gave to the profession his tuberculin and at once the hope was aroused that herein lay the long-desired means of cure. Numerous trials were made of this substance, but at length it was abandoned by the great bulk of phthisiotherapists as a means of treatment. This was followed in turn by Koch's tuberculin R. and again hopes were aroused, only to be dashed to the ground. Of the various antitubercle sera that were put on the market or were experimented with, none now survive or continue to attract attention save that made by Maragliano.

The chief benefit that grew out of all this investigation was the conviction in the minds of bacteriologists and pathologists that the true scientific method of combating this greatest of all human plagues lay not in any therapeutic agent but in its prevention. Moreover, the accomplishment of this lay in two directions, the eradication, or at least the very considerable diminution, of the possibility of infection on the one hand and the immunization of the human race on the other. Accordingly, men of the very highest scientific acumen and training like von Behring, Trudeau and a host of other workers in all lands are studying this question of prevention.

Of course, many questions arise in this connection which are fundamental. The universal prevalence of tubercle bacilli is recognized and it is known that infection may take place in many ways; but if any scheme of exterminating the disease is to prove effective it must be aimed at that most common source of the infection, human sputum. At the same time other less important ones must not be ignored. These and other topics connected with a preventive inoculation will be duly considered in another place.

Enough has here been said to point out the interesting and highly important turn in the line of research that has taken place since Koch's epoch-making announcement in 1882. We are as yet only on the threshold of labors calculated to prove the most salutary and far-reaching to mankind, and what is perhaps the most significant, is the fact that the public at large has been aroused and is actively coöperating with medical men in the attainment of that which, not long ago, might have seemed a Utopian dream.

Ætiology.—The discussion of this phase of pulmonary tuberculosis has not to do with the veritable cause of the disease, for this was settled beyond doubt by Robert Koch's announcement of the bacillus in 1882, but concerns all the manifold conditions which favor the entrance and growth of the germ in the human economy. Before entering upon the consideration of these, however, it will be well to state a few facts regarding the morphology and mode of growth of the organism.

Koch's announcement was all the more notable because it contained not merely a description of the germ but because it was so complete as regards the method of staining, modes of culture and experimental demonstration of its specific action that subsequent workers have been able to add nothing especially new or important to the facts therein stated. Baumgarten had recognized the bacillus but his methods for its recognition were crude and unsatisfactory, and hence it is only just to accredit Koch with the establishment of the important truths now universally accepted.

Morphology of the Bacillus.—The germ is (see Plate VII) a slender rod-like structure (hence the term bacillus) often slightly bent or curved, and having a length of 1.5 to 4 μ or one quarter to one half the diameter of a red blood-cell. When seen in properly stained sputum it is observed to be in clusters or in groups of two, united at one extremity at an obtuse angle, or joined end to end into a long slender thread. Some of the rods, furthermore, display breaks or gaps in their continuity which by some observers have been taken to indicate spore formation but by others are looked upon as a manifestation of a retrograde process. Occasionally the bacillus may show branching or clubbing which gives to it a certain resemblance to actinomyces; hence the suggestion that tuberculosis be classed among fungous diseases. Such deviations in form are found most commonly in the cultures, and are a result of modifying influences incident to their growth in artificial media.

Conditions of Growth and Destruction.—The tubercle bacillus may be cultivated in blood-serum, glycerine-agar, bouillon or on potato, and it grows best at the temperature of the human body, 37° C. to 38° C. or 98.6° F. to 100.4° F. It is destroyed by sunlight in from a few minutes to several hours, depending on its intensity, by decomposition in several days, and by cold. This last is not so active a destructive agent, however, as is heat, for Cornet saw bacilli retain their activity for a period of six weeks when subjected to a temperature of 14° F. above zero. When, on the contrary, tuberculous sputum was subjected to the action of steam it lost its virulence within a minute and under the influence of hot dry air in fifteen minutes.

Action of Chemicals.—Of great practical interest is the effect on sputum of certain chemicals usually relied on for destruction of the bacilli. Thus, when a quantity of sputum equal to that used in the experiments with heat was subjected to a 2-per-cent solution of corrosive sublimate, the germs were not destroyed after twenty-four hours, which doubtless must be attributed to the coagulating influence of the poison on the outer layer of the sputum. A 5-per-cent carbolic-acid solution was resisted for two hours, while by absolute alcohol the sputum was rendered inert only after twenty hours. The same length of time was required for destruction of the bacillus by a saturated solution of muriatic acid and a 3-per-cent solution of carbolic acid. The above facts are taken from Fraenkel and have an important bearing on the methods to be employed for the destruction of the expectoration from tuberculous subjects.

Prevalence of the Bacillus.—Concerning the ubiquity of the bacillus it is only necessary to state that it is found in practically all parts of the globe, though in some localities more abundantly than in others. Moreover, it is not peculiar to the human race but invades the tissues of practically all animals under suitable conditions, even the cold-blooded, as reptiles and turtles when in confinement. In fact no lesson is more impressive as respects the development of tuberculosis than the observation that wild animals which never have the disease in their natural state will often contract it when confined in menageries. Carnivora, as lions and tigers, are considered more refractory to tuberculosis than are the herbivora, and yet, when confined in cages, they not infrequently die of the disease. Monkeys and apes, rabbits and guinea-pigs are also said to be free from it in their natural state, but exposed to the infection in confinement they display a remarkable liability to it.

Domesticated animals are especially prone to tuberculosis although not all are equally liable. Thus horses seldom contract it, while dogs, cats and cattle seem particularly susceptible. Indeed, the percentage of tuberculosis among bovines is quite alarming when we consider their importance from the standpoint of human food. It is said that of the dairy stock of this country from 15 per cent to 30 per cent is tuberculous. Sheep are not often befallen, and yet are not immune. Among hogs the disease is said to be far more common than is generally supposed (Osler). A striking instance in point is furnished by the experience of a farmer who fed his swine with the milk from a tuberculous herd of cows with the result that they all became infected and had to be slaughtered. The disease is common also among birds and fowls.

The foregoing facts open up a fruitful theme for speculation. To what is the prevalence of tuberculosis among domesticated animals and household pets to be ascribed? Confinement is not the only factor: there must be infection and hence it is pertinent to inquire if conditions for infection be not furnished by their contact with man. Cattle undoubtedly infect each other when shut up in stalls; but swine, poultry and the like that get their food largely off the ground may readily become infected from bacilli cast out with human sputa. On the other hand, pigs and fowls are often fed on cow's milk or they roam at will in the stables occupied by cattle.

Intercommunicability of Human and Animal Tuberculosis.—Such queries raise the question, In how far can the disease be communicated from one kind of animal to another, from animals to man and from man to animals? This question is of especial importance when we consider the wid
use of cow's milk as an article of food for man, young children in par

The bacilli of bovine tuberculosis present some morphological diffe
from human bacilli but nevertheless have been believed infective to
beings. Conversely, bacilli of man were believed capable of transmiss
cattle. The world was therefore astounded at the statement made by
at the Congress for Tuberculosis at London in 1901 to the effect that

tuberculosis was not transmissible to man, and hence the elaborate precautions taken to guard against infection by milk or flesh of tuberculous cattle were unnecessary.

Elaborate investigations were at once undertaken in Europe and America to determine the validity of Koch's position. This is not the place to describe the manifold experiments that have been made and the arguments pro and con adduced by the various contestants. It must suffice to state that the matter is still not definitely settled. Theobald Smith in this country and many able experimenters abroad have successfully inoculated cattle with human tubercle bacilli, while various pathologists hold that the discovery of intestinal lesions in infants fed on cow's milk prove the susceptibility of humans to bovine tuberculosis.

Koch's supporters, on the other hand, maintain that the experiments going to prove the susceptibility of cattle to human tuberculosis are open to the charge of faulty technic and that even in the instances of intestinal tuberculosis in infants it is not certain that the infection was not of human instead of animal origin.

William Welch in an address delivered in Washington, D. C., at the first annual meeting of the Association for the Study and Prevention of Tuberculosis, May, 1905, stated that the transmissibility of human bacilli to cattle has been demonstrated beyond a doubt. Two points are yet to be determined, however, namely, the frequency in human beings of bacilli virulent to cattle and, of such cases, what percentage owed their origin to bovine bacilli. The communicability of bovine tuberculosis to the human cannot be proven experimentally and hence the determination of this phase of the subject must be left to the difficult and uncertain method of clinical observation. Light may be thrown on this question by the study of primary intestinal tuberculosis, but here we cannot always assert that the lesions are not due to human bacilli introduced through the mouth and derived from the handling by the infant of contaminated articles.

Modes of Invasion.—A question of prime importance in considering the ætiology of pulmonary tuberculosis concerns the manner in which the bacilli gain access to the human organism. It is generally admitted that this may take place in any one of four ways, namely, (1) by ingestion and hence along with the food, (2) by inhalation, (3) by trauma, that is, through the skin and (4) by direct or congenital inheritance.

(1) *By Ingestion.*—It is a generally accepted belief that tubercle bacilli frequently gain entrance to the human organism through the gastro-intestinal tract. Indeed this route of infection has been so abundantly proven by experimentation, i. e., by feeding young animals with tuberculous milk, that doubt of such an origin cannot be reasonably entertained. It is held to apply, however, mainly, if not exclusively, to the tuberculosis of intestines and mesenteric lymph glands seen in children, and either not at all, or but very exceptionally, to pulmonary tuberculosis, and on the ground that the bacilli cause disease at or in close proximity to the point of entrance.

Von Behring's View.—Very recently, however, von Behring has come forward as a champion of this mode of origin in practically all cases, even of pulmonary tuberculosis. He maintains that the disease is contracted in the early months of life as a consequence of feeding with contaminated cow's milk, and that the infection remains latent for a period of years. In the course of time, either when tissue resistance has been lowered by puberty or by depressing conditions, or in consequence of fresh infection, disease of the lungs declares itself.

In opposition to this view it is urged (1) that the transmission of bovine tuberculosis to human beings has not yet been satisfactorily proven. (2) If the intestinal tract were the atrium of infection evidence of the disease ought to be discovered in the mucosa of the intestines and such lesions have not been discovered. In reply von Behring asserts that in the first two months of extra-uterine existence the epithelial covering of the intestines is defective and hence that tubercle bacilli may pass through the intestinal wall and lodge in the neighboring lymph glands. This, he claims, has been abundantly proven by experiments on nursing animals.

In support of von Behring's contention that tubercle germs may pass through the intestinal mucosa without leaving a trace of their passage behind may be cited Ravenel's experiments. This investigator fed an emulsion of tubercle bacilli to fasting dogs and upon killing and examining the animals four hours later found the germs in the chyle and mesenteric glands but not in the walls of the intestines. Consequently we may accept as possible this route of entrance in some instances.

But the entire subject of primary intestinal tuberculosis has not been exhausted with the determination of the possibility of such an avenue of infection. In the first place, it is a question whether or not all of the instances observed in children are to be attributed to the ingestion of tuberculous milk. As will be seen in considering infection through the mouth and throat it is very likely that many of the cases owe their origin to human bacilli. As pointed out by William Welch in his Washington address, this question can only be answered by determining the character of the bacilli in each instance and this would involve an enormous amount of work.

Another important phase of this question relates to the frequency of primary intestinal lesions and their bearing on subsequent pulmonary phthisis. Welch is of the opinion that the incidence of this localization of the disease varies greatly in different localities, and hence accounts for the wide divergence of view regarding the frequency and importance of primary intestinal tuberculosis. The entire matter is intimately associated with the question of the transmissibility of bovine tuberculosis to human beings, but, as will be stated in considering the danger of feeding infants with cow's milk, statistics in England appear to show that the infant mortality has been
reduced since the Government instituted a rigid control of th
of Great Britain.

It is certainly the part of wisdom for health boards and
recognize the possibility of primary intestinal tuberculosis, be
use of contaminated milk and from the handling of babies b
mothers and nurses, in order that all necessary precautions

toward prophylaxis in individual instances. The mode of invasion next to be considered is by far the more important, but it does not for that reason minimize the danger of infection through the gastro-intestinal tract.

(2) *By Inhalation.*—Although the opponents of von Behring's theory, among whom may be mentioned Flügge, Fraenkel and Parrot, believe that infection through the food accounts for cases of tuberculosis of the intestines and intra-abdominal lymph glands, they do not believe pulmonary consumption so originates. In children the lungs become involved secondarily to tuberculosis of the bronchial glands. As these structures are too remote from the intestinal tract to render likely their infection through this route without coincident disease of the lymph nodes elsewhere, they believe the lungs become involved in consequence of the inhalation of bacilli along with respired air.

In the case of adults they believe the lungs are often, nay are generally, the seat of a primary infection contracted through inhalation. They recognize the natural protection afforded by the ciliated epithelium lining the air-passages and the fact that germs of all kinds are relatively scarce in the deeper parts of the bronchial tree. Nevertheless, they believe that either because this protective arrangement is defective through loss of the epithelia or injury to them, or on account of the entrance of bacilli in numbers so great as to overpower the protective apparatus, they may be lodged in the lungs and thus set up a local infection.

Sputum as a Source of Infection.—At all events, they are in accord with the majority of physicians who look upon the inhalation of dust impregnated with particles of dried tuberculous sputa as a very real source of danger and as responsible for many a case of pulmonary disease. It certainly would be very unwise to ignore such a possibility, and hence to neglect certain obvious precautionary measures growing out of such a belief.

There have been recorded numerous experiments and observations that go to attest the danger of respiring an atmosphere impregnated with tubercle germs. Thus, Straus detected bacilli in the nostrils of nine out of twenty-nine healthy persons whose duty it was to clean the apartments occupied by such invalids and to nurse them. There have also been numerous observations which prove that these minute organisms are carried in the fine spray thrown from the mouth in the act of coughing (Flügge, Laschtschensko, Heymann and others). The breath itself is not necessarily so laden, but the bacilli are contained in the invisible drops of moisture cast from the mouth. Accordingly, these observers have given one meter as the distance from the consumptive's body within which there may be said to exist a danger zone.

Dried tuberculous sputa are therefore a very prolific source of danger, and authorities now recognize that herein lies the most probable source of infection as regards mankind at large. Friends and attendants upon a person afflicted with pulmonary tuberculosis are, in addition, exposed to danger from impalpable sputum, i. e., from the bacilli ejected from the mouth in coughing, speaking, sneezing, etc. Nevertheless, it is the dust particles arising from the clothing, from the coverings of the bed, and from the floor and walls of the room which must be guarded against if one is not to contract the disease. It is this consideration which is responsible for the widespread crusade against spitting.

(3) *Infection through trauma,* i. e., abrasions of the skin, cuts, etc., on the part of persons in attendance upon tuberculous subjects has been proven more than once. True, such infections generally give rise to localized nodules which may be readily excised, yet, Gerber's experience with swollen axillary glands after such a mode of contamination goes far to prove that general and pulmonary infection may be the consequence. The likelihood of traumatic contraction of the disease may be remote, nevertheless the possibility is too real to be ignored.

(4) *Congenital transmission* of tuberculosis is urged by some, especially by Baumgarten. It has been shown by experimentation on guinea-pigs and observations upon a few cases in women that tubercle bacilli may be carried from the mother through the intervention of the placental tissues (Birch-Hirschfeld, Schmorl, Lehmann) or blood to the offspring *in utero.* There are no reliable observations going to prove direct transmission through the sperm of the male parent, although tuberculosis may be communicated to the mother in consequence of coitus when the male is suffering from tuberculosis of the genito-urinary tract.

Although the published instances of congenital transmission of tuberculosis from mother to fœtus are not numerous, they are yet sufficiently large to warrant the conclusion that this route of infection is not to be ignored. Indeed, William Welch, in his Washington address already mentioned, gave it as his opinion that Schmorl's studies indicate that the tuberculization of the fœtus from the mother is not so uncommon as might be supposed, and that hence physicians should not disregard such a possibility. It has an important bearing on the question not only of matrimony but also of child-bearing by tuberculous women.

However, that tuberculous disease is commonly or frequently inherited directly from a parent is not the case. It is, in short, too remote a possibility to be seriously considered in discussing the ætiology of pulmonary tuberculosis. The chief modes of infection are through the digestive tract, especially in contaminated cow's milk supplied to infants, and through aspiration of bacilli, usually in dust. In the case of adults the latter is probably the more frequent. We shall return to this in speaking of heredity.

A word may here be added with reference to the nursing of their babies by tuberculous mothers. Such should not be permitted, not because of danger of infection to the child through the maternal milk, for, according to von Behring, bacilli are practically never found in woman's milk, but on the ground that lactation is bad for the mother, and the clos
necessitated is a positive source of danger to the babe.

I have recently seen a six months' infant with tabes mes
mother was dying of pulmonary tuberculosis and who had be
five months of age. This does not invalidate my statement
garding the purity of mother's milk, for, as I apprehend, i
possible for the infection to have been conveyed to the chi

handling of the breast by the contaminated fingers of the mother. The danger of kissing must also be borne in mind in such a case.

(5) *Infection Through the Mouth and Throat.*—Westenhoeffer has suggested another very plausible source of infection in the case of young children. He believes that the time of danger is during the first dentition, when babies are allowed to sit and crawl upon the floor. He calls attention to the fact that at this time the gums are inflamed and sore, hence in a favorable state to absorb any germs that gain access to the mouth. If, now, the children are permitted to handle all sorts of articles picked up from the floor and to put them in their mouths, or if the mother who is herself tuberculous or who has been waiting upon a tuberculous member of the family rubs the child's swollen gums with her fingers, as is so often the habit, then, in some one of these ways, the bacilli may be readily taken into the mouth. From here they may be swallowed, or more likely they are taken into the lymph-channels whence they readily pass into the cervical, mediastinal and bronchial lymph nodes.

Another avenue of entrance is by way of the tonsils. These structures may be the seat of either primary or secondary tuberculosis, although the latter is much the more common. This matter has been well set forth in a recent paper by George B. Wood who believes, from clinical data, from reports in the literature and from *post-mortem* observations, that it may be safely asserted that "in cases of pulmonic phthisis secondary tuberculous infection takes place more readily in the tonsils than in any other part of the upper respiratory tract."

Thus, of 136 cases of pulmonary tuberculosis collected from various authors and examined with special reference to this point, the tonsils were found tuberculous in 94. It may also be stated that the liability to tonsillar infection is greater in acute than in chronic cases as shown by the observations of Schlesinger, who found these organs diseased in 12 out of 13 instances of galloping consumption. The reason for this lies in the enormous numbers of bacilli that pass over the tonsils in their passage outward along with the expectoration.

As showing the occasional occurrence of primary tuberculosis of the tonsils and of adenoid vegetations, may be cited Dieulafoy's experiments which he reported to the Academy. Two sets of guinea-pigs were inoculated with portions of these structures taken from apparently healthy individuals as follows: 61 with fragments of hypertrophied tonsils and 33 with pieces of adenoids. Of the former 8 or 13 per cent, and of the latter 7 or 20 per cent developed tuberculosis. From these results Dieulafoy concluded that tuberculous disease of the tonsils may exist without any other evidence of it than hypertrophy.

The bearing of this upon the development of pulmonary tuberculosis is to be found in the intimate connection existing between tuberculosis of the tonsils and cervical adenitis. The pharyngeal tonsils are lymphoid structures, and through their efferent vessels stand in close relation to the lymph nodes of the neck. The several chains of glands unite finally in the jugular trunk and thus pour their contents into the internal jugular vein on the right side, and on the left, either into the thoracic duct or in the vein, as on the right side.

If, now, the cervical glands become infected and ultimately break down, the bacilli thus set free, enter the venous circulation and are carried to the lungs causing miliary tuberculosis, as Wood found in all but one of the eighty-four animals experimented on. These facts are of immense importance as regards the danger from tuberculous glands of the neck, and also in explaining the terminal miliary tuberculosis in a certain number of cases of chronic disease of the lungs.

Primary tuberculous disease of the tonsils is far less common, but that it does occur is proven by the figures collected by Wood from the literature. Thus, of 1,671 cases in which these structures were examined with special reference to this point, evidence of primary tuberculous disease was found in 88 or 5.2 per cent. Tusseau, who has observed three instances of the kind, believes that infection may take place through the food, but to my mind it is more likely that the bacilli are introduced into the throat in some of the ways suggested by Westenhoeffer.

The consideration of the various facts that have just been discussed at length may be concluded by a translation of most of the conclusions to which Livius Furst has arrived, in his recent monograph, Die Intestinale Tuberculoseinfectionen mit besonderer Berücksichtigung des Kindesalters. Stuttgart, 1905. They probably represent the latest and most trustworthy views on the questions that are engaging the attention of pathologists and experimenters in this domain of medical science.

(1) An intestinal infection in children in consequence of the ingestion of bacilli contained in milk is not common but is possible, and, in all probability, proven.

(2) The possibility of an enterogenous, i. e., an alimentary tuberculous infection deserves attention in consequence of the exclusive nourishment by means of milk in the earliest portion of life.

(3) The proof of a primary intestinal tuberculosis must be left, in the first place, to pathological anatomy, but indirectly also to animal experimentation which has established the communicability of human tuberculosis to cattle.

(4) Statistics do not prove an exact parallelism between bovine and human tuberculosis nor a high percentage of primary intestinal tuberculosis.

(5) A primary intestinal tuberculosis, i. e., one due to ingestion, exists; but as compared with pulmonary tuberculosis of aërogenic origin, is far less frequent. Both modes of infection may take place in the same individual.

(6) It is possible for tuberculosis of the alimentary tract to occ
way of the lymphatics surrounding the upper part of the pharynx, a
this way it may give rise to a general or pulmonary tuberculosis.

(7) There can be no doubt of a predisposition as respects distribut
the disease, depending upon diminished local or constitutional resistan

(8) It has been proven that an intra-uterine infection with tuber
may take place through the placenta.

(9) It is possible for a virulent infection by either human or bovine tubercle bacillus to remain latent in the system for many years.

(10) Both these forms of bacillus are not fully identical but are to be regarded as nearly related varieties of an original form. The possibility of a reciprocal transference of the human type to cattle and of the bovine type to man has been fully established.

(11) The bovine bacillus attacks cattle more readily and infects them more strongly than does the human bacillus, while the reverse holds true of the tubercle bacillus as regards man.

(12) Cow's milk, especially mixed milk, often contains virulent bacilli of the bovine variety, particularly when there is disease of the udders or an open tuberculosis of the lungs.

(13) Cattle which failed to react positively to tuberculin may, nevertheless, be suspected of disease, and their milk may be dangerous from the presence of bacilli.

(14) Bacilli of bovine origin are capable of passing through the intestinal epithelium without leaving demonstrable lesions behind, and of becoming deposited in the follicles of the intestines.

(15) That process is to be regarded as a primary intestinal tuberculosis which, so far as this difficult question can be decided, is either the only or the oldest discoverable lesion.

(16) A tuberculosis of the mesenteric lymph nodes which exists without similar changes in the intestines, lungs or general system is, in all probability, one of alimentary origin.

(17) Undoubted cases of primary intestinal origin are certain although they are often an accidental discovery of the *post-mortem* room; they are few in number, have small tendency to propagation in the general system or to systemic infection, and represent but a small percentage of the children who, without becoming infected, consume uncooked or insufficiently sterilized milk.

(18) Although the two processes cannot be considered identical, bacteriologically, still the secondary intestinal tuberculosis of auto-infective origin which is seen in the phthisical, proves the possibility of an intestinal disease in children occasioned by the use of contaminated milk.

(19) The prevention of tuberculosis in childhood necessitates the following precautions: immunization of cattle, improvement of the condition of cattle in general, the production of pure milk, and the removal of all conditions favoring the communicability of tuberculosis from man to man.

Conditions Predisposing to Infection.—Since Koch's bacillus has been so conclusively shown to be the exciting cause of tuberculosis, and since we have seen how many and varied are the avenues through which the germ may enter the human organism, it only remains to consider such conditions as favor infection.

When we consider the great prevalence of tuberculosis and the notorious carelessness of individuals as regards prevention, it is not to be wondered at that one seventh of the human race dies of pulmonary consumption. It is remarkable rather, that still more persons do not succumb to this "great white plague." In accounting for the escape from tuberculosis of the large number of individuals who reach middle or advanced age and die of some other disease we must assume either that infection is less frequent than might be thought natural under the circumstances or that other conditions than mere infection are necessary if pulmonary disease is to result.

Regarding the *frequency of infection* it needs only to be stated that *post-mortem* observations have proven beyond doubt that very many more individuals are tuberculous than ever manifest clinical evidence thereof. Thus, for many years past the profession has been taught to believe that from 60 to 75 per cent of all persons dead by accident or other than tuberculous disease display some focus of this disease in the lungs or elsewhere. In 1899 O. Naegeli published in Virchow's Archiv the results of his research in this direction which furnished some very startling figures.

Thus, of 500 autopsies made by him in the pathological laboratory of Zürich he found evidence of tuberculous lesions in 100 per cent of persons over thirty years of age, in 96 per cent of those between eighteen and thirty, in 50 per cent of those between fourteen and eighteen, in 33 per cent of those between five and thirteen, in 17 per cent of those between one and five years. As stated by Fraenkel, Naegeli discovered lesions in 97 per cent of 284 adults.

It has been urged with reference to these statistics that the bodies examined came from the poorest classes which, as is well known, are especially exposed to tuberculous infection. In answer to this objection Fraenkel cites the necropsy records of Birch-Hirschfeld who, among 826 persons dead either from accident or from acute disease, found 171 instances of pulmonary tuberculosis, and yet, so far as known, these individuals had been free from clinical manifestations of this malady prior to death. In corroboration also of the prevalence of tuberculous infection among apparently well persons may here be cited the result of Franz's tuberculin injections.

This military surgeon tested the members of two Austrian regiments with tuberculin for the purpose of ascertaining how many were actually free from tuberculosis. These young soldiers are supposed to be among the healthiest members of society, and yet Franz found that 61 per cent of the men in the first year of service reacted to doses of 1 to 3 mg., while of those in their second year of service 68 per cent showed reaction to these small amounts. Had 10 mg. been injected he believes 100 per cent would have reacted.

The results obtained by Naegeli and Franz have been widely quoted and by many writers accepted as trustworthy evidence of the well-nigh universal prevalence of tuberculosis in the human. There are some, however, who regard the conclusions apparently to be drawn from their work as misleading.

Thus, in an editorial on "The Frequency of Tuberculosis" which appeared in the *Journal of the American Medical Association* of July 1, 1905, are given the objections advanced by Kraemer. His paper appeared in the *Zeitschrift für Hygiene*, 1905, and was not a presentation of new facts but an exhaustive criticism of the various figures that have been published from time to time.

In particular, Kraemer points out that the results of Naegeli's investigations and those of Burkhardt at Dresden, which agreed with Naegeli's at Zürich, are at variance with the figures obtained by Orth. This last-mentioned pathologist found evidence of tuberculosis in but 27 to 28 per cent of 1,087 autopsies. From these and other statistics cited by him Kraemer maintains that we should be very reserved in our acceptance of the doctrine of universal tuberculous infection.

It is urged by him that Naegeli's and Burkhardt's remarkably high percentages are to be attributed to several factors which do not necessarily obtain in the population at large. In the first place, Naegeli's material came from factory operatives who, as is well known, furnish an unusually high percentage of tuberculosis because of the inhalation of dust. In the second place, objection is made to the regarding of every suspicious cicatrix at the lung apex or other pathological lesion as necessarily of tuberculous origin. Finally, he objects to their conclusions on the ground that the material coming to a pathological institute in a densely crowded urban population does not fairly represent the conditions to be found in other, as rural, communities.

Regarding the results of Franz's tuberculin injections, Kraemer directs attention to the fact that, although soldiers are supposed to be drawn from the healthiest in the community, still, in the instance of the regiments selected by Franz, the test was hardly a fair one, since these two regiments had furnished an unusual number of tuberculous cases prior to the experiments.

On the whole, therefore, the conclusions to be drawn from Naegeli's researches may be set down as follows: Tuberculosis is so frequent an autopsy finding that it can be regarded as practically universal among all persons whose life-conditions are unhygienic; that it increases strikingly after the fifteenth year, or that age when individuals begin to follow a steady occupation and hence become exposed to infection; that well-nigh all persons of the poorer classes show evidence of tuberculosis if they reach advanced age, and that instead of pulmonary localization of this disease being rare in the old it is relatively very common in them. And in this last consideration is to be found a very cogent argument for the infectious rather than the inherited origin of this malady.

Individual Conditions.—The foregoing facts furnish convincing proof of the frequency of tuberculous infection and bring us at once to a consideration of the question, Why do some persons develop pulmonary tuberculosis while others exposed to and perhaps infected by the same virus do not die of consumption? In part, this query is answered by the fact that tuberculosis is not always of the lungs, but aside therefrom the explanation must be sought for in all those conditions of individual and social life which lessen tissue-resistance. The most important of these will now be discussed.

Heredity.—There is a widespread notion both in and out of the profession that some persons inherit a marked tendency to consumption, which tendency is so strong in some instances as to constitute a *tuberculous diathesis.* Accordingly, we find older writers dividing cases of phthisis into two groups, the innate and the acquired, and of these the innate or hereditary form was held to develop earlier in life and to furnish a worse prognosis. The belief in an inherited predisposition rests on the experience that the consumptive frequently gives a history of tuberculosis in some other member of his family, brothers, sisters, parents, grandparents, uncles, aunts or cousins.

Testimony in favor of a family tendency to the disease seems particularly strong when one learns that tuberculosis has befallen successive generations, especially if in a direct line, as grandparents and parents of the individual who, being of the third generation, is found tuberculous. So convincing did such facts in the anamnesis appear, especially at a time when no positive knowledge obtained concerning the cause of the disease, that we cannot wonder at the prevalence of this notion of heredity.

There was, however, no fixed limit to the inquiry, whether or not the family history should be made to include distant relatives, i. e., members of collateral branches, and hence wide variations exist in the statistics given by authors on this point of hereditary disposition. Accordingly, we find in the figures given by Cornet that Louis and Hischsprung reckoned it as 10 per cent, Hill, Leudet and others at 50 per cent, Solly at 55 per cent, Mayet at 70 per cent, and so on up to Rufz at 85 per cent.

Although it is a striking fact that certain families are particularly afflicted with consumption, still, in the light of present knowledge concerning the likelihood of infection through bacilli thrown off in the expectoration, there is coming to be an entirely different notion regarding this matter of heredity. Instead of regarding an inherited predisposition to pulmonary tuberculosis as an important ætiological factor, infection is now believed to explain the prevalence of phthisis among various members of the same family circle.

Upholders of this view point to the intimate social relations existing in the home and to the liability that the disease may be communicated from parent to child, from child to child, and even from child to parent. Respecting this last possibility Cornet gives the following impressive figures. Out of 751 families in which the oldest child was the first to become tuberculous there died within the first ten years following the birth of the child 40 fathers and 38 mothers, while after the tenth year 125 fathers and 139 mothers succumbed to the disease. All these parents had been healthy prior to the birth of the first tuberculous child. These fathers and mothers could not be said to have inherited consumption from their own offspring, hence the inference is justifiable that they acquired it by contact with their children.

In past times, before the doctrine of prevention was taught, no pains were taken to destroy the sputum, the consumptive's room or bed was shared by some other member of the family, and in many instances the same house was occupied by one generation after another. Under such circumstances it was not at all strange that infection should take place, indeed it would have been most strange had the disease not been communicated from one to another. Even to-day, in spite of the cry that is being everywhere raised concerning the communicability of the disease, there are ignorant families in which the important laws of prevention are violated. But even where the invalid and his friends think they are exercising due precaution they are, in reality, not sufficiently careful, and hence the conditions for infection are present.

Only yesterday I saw a young man of eighteen years in an advanced stage of consumption whose mother declared there had been no instance of the disease in his family. On further inquiry, however, it was ascertained that he had resided in the same house with his sister-in-law who had died of tuberculosis. Heredity cannot be claimed in this instance, but judging from the conditions found at the time of my visit I am driven to the assumption of direct infection as the ætiological element.

Natural Lack of Tissue-resistance.—Whereas I do not any longer believe in congenital predisposition (i. e., tuberculous diathesis) as the main or even an important ætiological factor, I do yet believe that not all individuals are equally prone to pulmonary tuberculosis. In other words, I believe that some individuals display a manifest want of ability on the part of their systems to resist the infective agent after it has once gained entrance. When we look at the small chests, broad intercostal spaces and drooping shoulders of many of our tuberculous patients, at their delicate skin and slender bones, when we note their evident delicacy of constitution and obtain a history of symptoms which point unmistakably to neurosis, shown by erethism or by unnatural abhorrence of meat, fat, etc., etc., when we take all these peculiarities into consideration together with an absence of tuberculosis in the family history, it seems to me we cannot escape the impression that such individuals possess an inherent delicacy of structure quite different from many another sturdy specimen of humanity. It is as if tuberculosis finds ready victims among the highly organized, sensitive persons whose families are so intellectual and refined that they run all to spirit and brain and are physically incapable of coping successfully with this practical material age. They have become refined to a degree which makes them especially lovable and attractive, but unfits them for this rough world.

The advocates of the infection theory pure and simple may argue that this delicacy of constitution is not congenital but is the result and expression of a tuberculous infection that, acquired, perhaps, in infancy, has yet remained latent. Such a view would certainly be in accord with von Behring's doctrine; but, whereas the possibility of such a state of things cannot be denied, I still cling to the view that by reason of an inherited or congenital weakness of constitution the descendants of tuberculous forebears are more liable to develop pulmonary phthisis than are children of a more rugged stock.

Faulty Chest-formation.—Although not prepared to assert that a faulty formation of the thorax is a leading factor in the development of pulmonary tuberculosis, still it may be regarded as an element of weakness by hindering the proper expansion or ventilation of the lung. Since ancient times physicians have been accustomed to characterize the chest indicative of a tuberculous diathesis as the long, thin, flat chest, narrow at the apices and with broad ribs and wide intercostal spaces. It is rather startling, therefore, when Woods Hutchinson comes forward with the statement, and with figures to back up his statement, that the typical thorax of the tuberculous is one which instead of being flatter than normal, i. e., shorter in its dorso-ventral diameter, is in reality rounder than it should be; in other words, is one in which the natural rotundity of early childhood has been retained. Hutchinson finds that the normal relation in the adult is 70 from front to back as to 100 transversely and says: "From the evidence so far presented, the following conclusions seem to me reasonably probable, though, as before stated, I do not feel justified in advancing any more positive position than this until I have secured at least 500, and preferably 1,000, measurements taken on all classes and nationalities:

"First.—The typical tuberculous chest is round instead of flat and has an average index of about 80, nearly 10 degrees above the normal.

"Second.—This type of chest precedes the disease.

"Third.—It is an abnormal persistence of the fœtal, infantile and child type of the chest.

"Fourth.—Any chest more than eighteen years of age which shows an index of 80 or higher should be regarded as abnormal and as rendering its possessor more than usually liable to tuberculosis.

"Fifth.—The occurrence of such a chest in any patient over eighteen years of age, suspected of tuberculosis, raises a strong probability of the disease.

"Sixth.—The chests of growing boys and girls should be systematically measured at stated intervals, and whenever the index is found distinctly higher than that normal for their age active measures should be taken to remedy the defect.

"Seventh.—All those sports and exercises which involve wide-swinging use and play for the arms, chest, shoulder group of muscles, such as tree-climbing, swinging from ladders, from rings, from bars, ball-throwing, spear-hurling, tennis, swimming, will tend to correct this defect and flatten the chest down to its proper index.

"These, in fact, are the influences which have made the human chest shape and all that is needed to perfect it in the individual is a healthy reversion to the arboreal habits of our prehuman and the war sports of our savage ancestors." (See *Journal of the Am. Med. Ass.*, vol. xl, p. 1196, April 25, 1903.)

Whether Hutchinson's views shall prove correct or not remains to be seen. My own observations have been too few to permit me to draw trustworthy conclusions. In a number of instances I found his statements correct, but in others the contour of the chest was not infantile. The rule is not invariable, and in how large a proportion it does obtain is yet to be worked out. It remains to be determined why the chest retains this shape in some persons and not in others.

Defective ventilation of the lungs is certainly a factor of no mean portance. The apex of the lung is the portion in which tuberculous l first make their appearance and attempts have been made, therefore, account for this greater vulnerability on the part of the apices. It has b explained on the ground of lessened blood-supply (relative anæmia), so as owing to the deficient ventilation of this as compared with o ts of the lung. This, according to Birch-Hirschfeld, is accounted for the anatomic arrangement of the bronchus leading to the apex, it being given off at a sharp angle and hence tending to interfere with complete expansion of the lobules situated at the apex.

It is highly important, therefore, that children be corrected in any habits of posture that interfere with proper expansion of their chests, and that they early receive adequate treatment for nasal catarrh, adenoids or any other condition conducive to mouth-breathing. The thorax of the habitual mouth-breather is misshapen, being relatively too deep in its antero-posterior diameter at the base and too contracted at the apices. Moreover, the child or the adult whose mouth is kept open fails to make use of the protection furnished by nature in the nares against the inhalation of dust and bacteria. Furthermore, the unhealthy state of the nasal mucosa seen in sufferers from catarrh supplies conditions favorable to the growth of whatever germs, e. g., tubercle bacilli, lodge in the nostrils, and from here they can readily pass into the submaxillary lymph nodes, whence also into the mediastinal and bronchial glands.

Parents and teachers should see to it that the children committed to their charge should stand and sit erect, and not crouch over their books at desks entirely too low for them. As myopia tends to make children stoop, such a visual defect should early be recognized and corrected. Stooped shoulders are frequently the result of weak back muscles, and hence such youngsters are often straightened by appropriate physical exercises. Only this past Summer I discovered impaired resonance at the left apex of an eleven-year-old boy which arose from a bad habit of stooping. Systematic breathing exercises at length restored the apex to normal pulmonary resonance. Had it been allowed to go on it might, in time, have resulted in permanent lack of expansion and in possible pulmonary disease.

Age.—Individuals fall victims to tuberculosis at all ages, but as regards its localization in the lungs there are striking differences. Thus, although young children may have pulmonary consumption as a part of a general infection they are especially liable to glandular and bone tuberculosis. This is not quite easy of explanation unless it be on account of the greater susceptibility of the gastro-intestinal tract and their greater liability to contamination by tuberculous milk. The lymphatic system is relatively active in children and this is thought to account for the predominance of glandular tuberculosis in them. In the view of some, even when the lungs become affected, it is secondary to disease of the bronchial glands. Perhaps the reason children do not oftener succumb to pulmonary tuberculosis is because they die of the intestinal or mesenteric infection before time is afforded for the lungs to manifest gross clinical changes. Below the age of five years tuberculosis in the lungs appears as a miliary tuberculosis, whereas from that time on there is an increasing liability to pulmonary consumption in the form in which it is seen in adults.

There is a general belief that this disease is more prevalent in early adult life than at either extreme. West states very positively that there is a greater prevalence of the disease (66 per cent) between the ages of twenty and forty, having drawn his conclusions from his out-patient service. Cornet, who looks upon statistics based on small numbers as highly misleading an who has compiled extensive tables taken from the records of the Prussian Government, holds a contrary view to that of West.

Cornet's figures show plainly that although tuberculosis may befall a greater total number of persons in the middle period of life because of the greater number of individuals living at that age, still, when the number of tuberculous as compared with nontuberculous is computed with respect of age, the disease exists in a far higher relative percentage among persons past sixty years. In fact, this pulmonary disease increases in relative frequency with every quinquennium after fifteen. In this respect the tables of Cornet agree with Naegeli's *post-mortem* records previously given; from one to eight the percentage was 17, whereas from thirty on it was 100. The only conclusion to be drawn from all such figures is that as we advance in age we are increasingly exposed to infection. The aged may, by reason of lowered vitality become more ready victims, but it seems to me this question of the influence of age cannot be divorced from that of infection.

Indeed, as we reflect upon this subject of the ætiology of pulmonary tuberculosis, we see that it resolves itself into a mere question of seed, soil and suitable conditions of growth. The seed (bacilli) abound on all hands, while human beings furnish an ever-present soil or culture medium. Given unhealthy and unsanitary life-conditions, the seed will spring into activity and at length burst forth as the fully matured plant which we call tuberculosis. Render the soil unfavorable to the maturity of the seed by placing it in the most hygienic environment possible and it will prove barren ground and the unwholesome seed will perish.

Sex.—The influence of sex upon the mortality from consumption cannot, in so far as such exists, be attributed to the peculiarity of sex *per se*, but to conditions of life intimately associated therewith. Cornet's figures show a preponderance of tuberculosis among girls from the second to the twentieth year, whereas from the latter age on males are more often befallen. These differences probably depend, as Cornet states, upon the manner of life of the two sexes. During childhood girls are more closely confined to the house and are brought into more intimate association with the mother, who may be tuberculous. Boys during this period are given to play in the open air and hence are less exposed to infection.

When, however, males reach the age at which they begin work in factories and at various trades, their liability to infection increases, and with it they display a greater death rate from pulmonary tuberculosis. Consequently, these and other considerations make it plain that in sex alone cannot be sought an ætiological factor.

Pregnancy.—That in some cases a close relationship exists between child-bearing and the disease now considered is a matter of common observation. The reason, therefore, is probably to be found in the effect of pregnancy upon the general health of the woman. Some women seem to find in pregnancy a natural stimulus to improved nutrition and hence grow well and strong. Others find it a drain on their nutrition which they can ill endure, especially if they bear children frequently.

When pulmonary tuberculosis develops during or immediately following pregnancy it is probably on account of the lessened resistance to infection thereby engendered, or because in pregnancy is given a condition favorable to the manifestation of a preëxisting but latent tuberculosis. Hanau is said by Fraenkel to hold the opinion that, in consequence of the physical exertion of gestation there may be set up an auto-infection due to aspiration of bacilli from an old tuberculous focus in the lung, and that this may account for the not infrequent development of phthisis florida after childbirth.

Lactation.—It is not uncommon for a nursing mother to develop signs of pulmonary tuberculosis during this period of her motherhood. It is not unlikely that in some instances the outbreak of the disease is explicable on Hanau's hypothesis, but in others the health fails at too late a period to ascribe it to the mechanical effect of delivery in the manner claimed by Hanau. It seems to me far more likely to be due to the weakening effect of the pregnancy or of the lactation itself. Far be it from my intention to discourage young mothers from nursing their offspring, and yet if such have a tuberculous taint they should not attempt lactation or else they should pay strict attention to the maintenance of their nutrition and should refrain from social dissipation and aught else that may still further impair their strength. The bearing and nursing of children at too frequent intervals, as well as too prolonged lactation may certainly act as predisposing conditions by lowering resistance to infection.

Lack of Sufficient or Proper Food.—This is a great contributing factor in the ætiology of the disease in question, but in the case of the poor can hardly be considered apart from other conditions connected with their poverty. In the case of the well-to-do, however, want of nourishment is sometimes an important factor. Growing children sometimes take far less nourishment than their active bodies require. This may be due to some gastric disorder, but in other instances results from bad habits, the child being more fond of cake and sweets than of wholesome nutritious articles of diet. I have seen more than one tuberculous girl whose mother stated that all her life she was a poor eater, and hence, in my opinion, she had seriously undermined her powers of resistance to infection. This is not a trivial matter in the case of the individual who gives a history of consumption in the family.

Anæmia is another predisposing factor closely associated with the one just considered, since it may be the direct consequence of deficient nutrition. However, the anæmia referred to is that secondary to loss of blood from whatever cause. Single hæmorrhages may be recovered from without the individual's becoming thereby a prey to consumption, but chronic anæmia, such as is seen in young women from too profuse menstruation, etc., is a very different matter. In this connection it may be stated that there is a form of chlorosis intimately associated with the development of pulmonary tuberculosis. Whether it is a manifestation of the tuberculous infection or is but a contributing element is to my mind not fully established. Nevertheless, it seems to me to emphasize what has been already said concerning the danger to young people of an impoverished blood-state. Anæmia diminishes vital resistance, and whatever does that increases the danger of pulmonary tuberculosis.

Hæmoptysis.—In connection with what has been said concerning loss of blood, it may be well to consider the relation existing between pulmonary hæmorrhage and the development of consumption. By older writers, as Hoffmann, Boerhave, van Swieten and Niemeyer it was believed that hæmoptysis was a direct cause of the disease. Such a view is now no longer tenable in the light of present knowledge concerning infection. The hæmoptysis is a manifestation of pulmonary tuberculosis and is probably a result of rupture of a vessel-wall or of thrombosis in consequence of invasion of the vascular coats by tubercle. When, as sometimes occurs, it is followed by diffuse tuberculous broncho-pneumonia, we may assume that the hæmorrhage has carried with it into deeper parts bacilli which have then excited an exudative and proliferative inflammation. In other words, the outbreak of the cheesy pneumonia is the result of aspiration of the tuberculous germ in the same way as when bacilli are discharged into the bronchi from a caseous bronchial gland or from the breaking down of a cheesy focus in the lung.

Dissipated Habits.—Abuse of alcohol and all other forms of excess or riotous living which undermine the health are powerful factors in the development of pulmonary tuberculosis. This is attested by the frequency of the disease among hard drinkers and prostitutes. Their vices make them lead irregular lives and, in the end, reduce them to squalor. Their haunts are not only haunts of vice but are situated in dark unwholesome and overcrowded alleys and byways where infection lurks on every hand. It is not a mere figure of speech that vice loves darkness. It is literally true, and in dark damp cellars is the "stronghold of tuberculosis." Therefore, drunkenness and vice are contributing factors because, besides sapping vitality, they expose their votaries to infection.

Conditions of Life and Environment in General.—Under the preceding headings have been considered sundry predisposing factors residing in the constitutional peculiarities, necessities and habits of the individual, as we see them in tuberculous patients belonging, many times, to the better classes of society. We must now discuss certain ætiological elements as they concern people in general. These, as will be seen, are by far the most important factors in maintaining the prevalence of this dread disease, and it is against these that the efforts of sanitarians should be and are chiefly directed.

The Tenement.—Pulmonary tuberculosis is by no means confined to the poor, but its fearful ravages are more apparent among them because, on account of their poverty and ignorance, they are especially exposed to infection. In great cities like New York they are compelled to live in tenements which, on account of the greed of landlords, violate all the laws of sanitation Rotten old buildings are made up of narrow dark hallways, foul rick stairways and small dark rooms which at best are lighted by one or t narrow windows that open into an air shaft but a few feet wide and 50 or feet deep. Many of these closets in which human beings are forced to l are only 6 by 7 or 8 feet in size and have no window at all, or but a sin apology for one, situated high up and barred to prevent intruders fr crawling in from across the two-foot wide air shaft

These wretched abodes reek with filth and are choked with dust that has been years in accumulating. In Winter doors and windows are kept closed to exclude the cold, while in Summer hot, stifling air ascends from the dank foul cellars and cannot escape on account of the lack of skylights in the roof. Consumptive tenants inhabit the miserably gloomy, ill-ventilated rooms for months or years, infect the walls and floors with myriads of bacilli that, held in the dust and dirt retain their virulence, sometimes as long as two years. Never thoroughly cleaned and disinfected, these rooms forever receive accessions of new tenants after the death and removal of the old ones. Not infrequently these in turn become tuberculous and by their expectoration add to the infectiveness of the place, so that, as is well known, the previously healthy members of successive families become stricken with the plague and die to make room for fresh victims.

In New York City there are 361,000 such dark undersized rooms as have been inadequately described. From a single block of overcrowded tenements on the East Side known as "The Lung Block" there were reported in the ten years ending 1900, 265 cases of death from consumption and it is believed that for one reason and another not half the actual number was sent in to the City Health Department. In the First Annual Report of the Tuberculosis Commission of the City of New York, from which the above facts were taken, numerous instances are cited of apartments made up of two small, dark rooms which have infected family after family for a period of five or seven years.

The state of things in Chicago is not quite so bad, and yet the records kept by the Chicago Society for the Prevention of Tuberculosis show many instances of families of six or more members living in similar tenements, and of many cases of consumption among such ignorant, poverty-stricken families. The sick one, unable to rise or care for himself, lies in a dirty bed in the same room where the others live, sleep and eat, and in nearly every case his infected bed is shared at night by one or more young, susceptible children. In Chicago, attempts are made by the City Health Department to get such rooms cleaned and disinfected before fresh tenants are permitted to move in. But instances are only too many of houses from which cases of pulmonary tuberculosis have been *annually reported*. The conditions directly responsible for the prevalence of this dread disease among the dwellers in tenement districts will be discussed a little later on.

It must not be supposed that infection is limited to these wretched occupants of such unsanitary abodes. By no means! Most of the tenants are victims of the sweatshop system. Either in shops as dirty and unwholesome as their homes, or in their own forlorn homes, the poor wretches earning but a few paltry dollars a week labor over garments day after day, coughing and spraying upon their work the germs of the disease which is wasting their bodies to skeletons. These infected clothes are then sold in shops to thousands of susceptible individuals who are thus exposed to the dangers of consumption. In this and in countless other ways the infection is spread among the poor and from them to dwellers in other more favored localities.

The horrors of life in the tenements and the perils to society arising therefrom have not been exaggerated. On the contrary the picture has not been drawn in all its ghastliness. This needs the experience of the eye-witness, and that the social conditions of the poor beings who fall victims to consumption may be duly set forth I will quote from a thrilling address: The Plague in its Stronghold: Tuberculosis in the New York Tenement. Ernest Poole. (Taken from the report of the committee mentioned above.)

"At 18 Clinton Street, back in the rear tenement, a young Roumanian Jew lay dying of consumption. I had come in with a Jewish doctor. With every breath I felt the heavy, foul odor from poverty, ignorance, filth, disease. In this room ten feet square six people lay on the floor packed close, rubbing the heavy sleep from tired eyes and staring at us dumbly. Two small windows gave them air, from a noisome court—a pit twenty feet across and five floors deep. The other room was only a closet six feet by seven, with a grated window high up opening on an air shaft eighteen inches wide. And in that closet four more were sleeping, three on a bed, one in a cradle.

"The man's disease was infectious; and yet for two long weeks he had lain here dying. From his soiled bed he could touch the one table, where the two families ate; the cooking stove was but six feet from him; the cupboard over his pillow; he could even reach one of the cradles, where his baby girl lay staring, frightened at his strange position: for his wasted body was too feeble to rise; too choked, too tortured, to lie down. His young wife held him up while the sleepers stared silently on, and that Yiddish whisper came over and over again, but now with a new and more fearful meaning: 'Breath—breath—breath! Or kill me; oh, kill me!'

"Two years ago this man had come to America—one of the 488,000 in 1901. He came young and well and hopeful, with his wife and their baby son. Two more had been born since then. It was to be a new country, a new home, a fresh start, a land to breathe in. 'Breath—breath—give me breath!' He had breathed no air here but the close, heavy air of the sweatshop, from six in the morning until ten at night. Sometimes—he whispered—he worked on until eleven. He was not alone. In New York to-day and to-night are over 50,000 like him, working. And late in the night when he left his feverish labor, at the hour when other homes are sleeping, he had come in through the foul court and had sunk into a restless sleep in the dark closet six feet by seven. There are 361,000 such closets in the city. And this was his home!"

The foregoing is not the picture of a single instance, but graphically portrays the misery, poverty and industry of the wretched human beings who swell the vast army annually sacrificed to the plague, consumption. The conditions of their daily life are the same, only in an aggravated form, which elsewhere maintain and spread this terrible disease. In a word, the conditions are such as keep alive tubercle bacilli for months and years, and, by undermining the health of the poor beings forced to endure them, prepare the soil for the growth of the germs in their lungs. These conditions will now be considered in detail.

Overcrowding.—Of all the social factors contributing to the ætiology of this mighty scourge, no other is so important as overcrowding or the herding together of large numbers of individuals in quarters wholly inadequate for their suitable accommodation. This is shown not only in tenements but also in prisons, barracks, convents and the like, since in such institutions the death rate from phthisis is invariably higher than among an equal number of persons spaciously housed. When families of eight or ten are compelled to dress, eat and sleep in two small rooms and to breathe over and over again the same atmosphere never entirely replaced by fresh, pure air from without, all conditions for infection are provided.

Sanitary laws cannot be enforced, proper cleanliness cannot be maintained, filth accumulates in the corners, dust collects on the walls, and tubercle bacilli that have gained entrance float in the atmosphere. The health of the inmates becomes impaired and is still further reduced by lack of nourishing food. Vice and drunkenness add their share to the evils of privation, or long hours of grinding toil make impossible recreation in the fresh air of parks and country. Children grow up pale and stunted and their parents grow old before their time. In short, they are rendered unable to resist disease.

Infection lurks on every hand and only those persons escape or resist it who are the most robust. Accordingly, the prevalence of consumption may be said to be directly proportionate to the density of the population. In all large cities it is most rife in the wards and blocks that are the most crowded. The death rate from this disease is said to be twice as high in cities of over 20,000 as it is in towns of less population. Maps prepared by the Chicago Society for the Prevention of Tuberculosis and by Dr. T. S. Sachs show in an impressive manner the direct relationship existing between this scourge and the density of the population, some blocks in the Ghetto being veritable plague spots.

Impure air is unavoidable in overcrowded rooms and houses, and is the great medium for spread of the infection, but it is not confined to the homes of the poor. Indeed, on all sides, in the residences of the well-to-do, in street cars, workshops, churches, places of amusement, offices, stores, in short, wherever people abound there is a lamentable lack of proper ventilation. Wherever persons congregate some may be heard to cough, and here and there some filthy or thoughtless individual expectorates on the floor. Dust is stirred up and, carrying bacilli perchance, is inhaled by beings whose lungs offer an excellent soil for their growth. *Pulmonary tuberculosis is a house-born disease*, and yet the laity and the profession are indifferent to the crying need for fresh air in homes and public buildings.

Public conveyances seem to me a great source of danger, since they are overcrowded, notoriously ill-ventilated, and carry as passengers the sick and the healthy, the tuberculous and the nontuberculous. If the consumptive does not actually expectorate on the floor, yet, when he coughs, he sprays bacilli into the atmosphere and perchance onto the clothing or into the face of some fellow passenger. With doors and windows shut tight, the ventilators closed and not a current of fresh air to drive out germs, our street cars cannot fail to be carriers of infection.

Office buildings, large department stores and other public structures are many of them provided with odious revolving doors intended to shut out dangerous draughts of cold air. As a matter of fact they serve to seal hermetically the hallways against the ingress of pure air, and thus compel employees to breathe over and over again, during ten or twelve hours, an atmosphere charged with death-dealing germs among which are tubercle bacilli. Until about a year ago a certain wholesale hardware firm in Chicago compelled its hundreds of clerks to spend their days, Summer and Winter alike, in a deadly atmosphere because windows were kept closed tight to shut out dust and smoke. A method of ventilation was provided but was utterly inadequate, and in this den, tuberculous persons worked alongside of other clerks, as I knew from my certain knowledge.

Hundreds of families among people who ought to know better make it a practice to sleep with windows closed. The night air is considered dangerous, and yet the peril from germs is disregarded even though some member of the household may have consumption, or the house may have been previously occupied by a tuberculous tenant. In these and in other manifold ways children and delicate persons are daily, nay hourly, exposed to the liability of infection. If the air be not so foul as in the homes of the tenement dwellers, it is yet impure and a menace to health.

Lack of sunshine is another exceedingly potent element in the perpetuation and spread of pulmonary tuberculosis. When we reflect on the ease with which sunlight can destroy the bacilli and on the fact that hundreds of thousands of homes in our great centers of population never have a ray of sunshine penetrate their dark recesses, it should be regarded as a blot on our boasted civilization. The same thing holds true of the factories, shops and basements in which young girls and boys are employed. They cannot be expected to keep their health, to have vigorous bodies and sound lungs when forced to breathe foul air in rooms never purified by this natural means of defense against disease-bearing germs. But even in homes situated where this boon to humanity can enter, it is only too often excluded by window shades and heavy draperies lest it fade furniture or rugs, so that health is made subordinate to home decoration.

I know of a home in Philadelphia where the father and three sons all died of this "great white plague" and where the mother carefully shut out the sunshine. After every death, in accordance with an old custom, the inside shutters were tied with black ribbons in token of mourning for the dead and utter obliviousness of the welfare of the living. Such things are abominable. Yet, as long as greed, on the one hand, shuts out sunlight from the tenements of the poor, and ignorance or a mistaken sense of the fitness of things, on the other, continues to exclude the rays of the beneficent sun from the homes of the wealthy, pulmonary tuberculosis must be expected to decimate the ranks of human beings.

Dampness is the fourth great factor in maintaining infection. It
no difference whether it be owing to defective drainage, as was pro
the historic investigations of the late Henry Y. Bowditch, or to w
sunshine and fresh air, or to having the house surrounded by tre
consequences are the same. Dampness of the walls but intensifies t
lurking in the dust and prolongs the life, *not of the inhabitants, bu*

bacilli. Overcrowding, foul air, want of sunshine, and dampness form a deadly quartet which, as long as they are permitted to dominate the homes of our masses, are bound to furnish victims for this terrible monster that is annually destroying millions of human beings.

The proof of their potency is found in the experience of the large cities of England. Formerly, to a far greater extent than now is the case, there were in English cities tenement districts in which rotten old buildings were huddled together on either side of narrow, dark alleys and byways. Filthy, foul-smelling and dark, they were thronged with thousands of beings who never saw a ray of sunshine nor breathed a breath of pure air. Bacilli reveled in their forbidding recesses and fattened on the poor wretches doomed to spend their lives in these haunts of misery and vice. At length, such was the mortality from tuberculosis in these wretched abodes that steps were taken to replace them by modern, well-lighted and ventilated tenements, with the result that the death rate from consumption has been materially reduced.

The effect of unsanitary surroundings has been shown by the high mortality from this disease in prisons, barracks and homes of religious orders. In prisons most of the unhealthful conditions above described prevailed formerly more than at present, and yet, even to-day, pulmonary tuberculosis is far more prevalent in them than outside.

West states that Baly in his report upon Millbank Prison showed that the death rate from phthisis was more than three times that of the general population, being 43 as compared with 13 per cent. Of two Austrian prisons cited by Parkes in illustration of the effect of ventilation, the mortality in the one well ventilated was 7.9 per cent and in the other without adequate supply of fresh air it was 51.4 per cent. In a Wuertemberg prison mentioned by Cless the death rate from consumption between the years 1850 and 1859 was 24 per cent, whereas by improved sanitation it became reduced to 8 per cent, the latter rate being still, as remarked by West, three times that of the general population.

It is stated that among soldiers the death rate from pulmonary tuberculosis is greater in times of peace than of war. This is attributed to their confinement in barracks during peace, whereas during war they live and sleep out of doors. West states that in Canada the mortality among soldiers confined to barracks was 23 per 1,000 during the years 1830 to 1837, whereas after the barracks were drained and ventilated it ultimately fell to 6 per 1,000.

Cornet makes some startling statements concerning the mortality from tuberculosis among the nursing sisterhoods of Prussia. Thus, whereas among the people at large it was from 15 to 20 per cent of the total death rate, among the nuns of the nursing communities it reached the terrible figure of nearly 63 per cent. The average age at death was 36.27, so that although the sisters were in good health when admitted to the orders, still their life-expectancy at twenty-five was only eighteen years, or the same as that of ordinary females at fifty-eight.

In Chicago especial attention has been called to the frequency with which cases of consumption have been reported from lodging houses in the down-town district. Comparative statistics are not to be obtained, but some idea of the danger lurking in men's lodging houses may be had from the fact that within the thirteen months preceding January 1, 1905, there have been 10 cases reported from a single house, No. 134 Madison Street. Also, out of 4,561 patients admitted to Cook County Consumptive Hospital between November 1, 1899, and February 11, 1905, 2 per cent have come from three lodging houses, 42 cases from a single lodging house. In these cheap lodging houses practically all the conditions for the maintenance and spread of the infection are present. Relatively large numbers of men sleep in bunks arranged in tiers around all sides of a common room or in tiny rooms divided from one another by thin wooden partitions; sunlight rarely or never enters; men with coughs herd with many others and are careless in the disposal of their sputa; ventilation is imperfect and, although not filthy, these houses cannot, from the very nature of things, be free from infected dust. Many of the inmates are broken in health by reason of drink or poverty. These cheap places of habitation are for the most part not so objectionable as to receive condemnation by the city health authorities, and yet they exemplify the social factors in the causation and distribution of this widespread disease.

Occupation.—In occupation *per se* there cannot be said to reside a causative factor of pulmonary tuberculosis. An employment necessitating the production and inhalation of dust is injurious to the respiratory organs as shown in the chapter on Pneumonoconiosis, but it does not always give rise to tuberculous phthisis. One that involves bending over machines or workbenches all day long may ultimately prove harmful by creating the habit of stooping and hence deficient ventilation of the lungs, but when an occupation favors the development of consumption, it is because of conditions connected with the factory and the long hours of work rather than with the nature of the work itself.

Thus, it must of necessity be most injurious to health for half-starved wretches to work in sweatshops from twelve to sixteen hours at a stretch, the size of the shop being entirely too small to supply even the lowest possible air space allowable for the workers, and then not provided with proper means for ventilation. The sweatshop not only unfits its victim to resist the infection waiting for him in his unsanitary home in the tenement, but it exposes him to bacilli coughed out by his equally wretched fellow laborers.

Thus far we have been considering the *danger to the individual* engaged in such occupations, and in a strict sense such is the main consideration that should be given to the nature of a man's work. In a broader sense, however, there is an intimate relationship existing between occupation and the spread of tuberculosis; namely, the *influence of the sweatshop in spreading this infection through the community*. The instance of the Roumanian Jew who worked on for weary weeks and months after disease had fastened upon hi
was no fancied picture.

Hundreds of consumptive men and women are to-day and every
sewing on clothing which is to be worn by other hundreds of tender child-
These garments are infected and capable of carrying bacilli to their wear
Thus this greatest of all plagues is spread through the community and

ever be, as long as the sweatshop is permitted to exist, or as long as miserable half-starved human slaves-to-greed are compelled to live in tenements that would not be considered fit for the horses of the rich. Brave-hearted, philanthropic men and women are battling against this terrible disease, but their efforts will be in vain as long as vile tenements and sweatshops are tolerated in our midst.

Nationality.—As with occupations, there is nothing in race or nationality directly responsible for the prevalence of pulmonary tuberculosis. All peoples, whether white, black, brown or yellow, are equally liable to contract the disease when equally exposed. The Jews have been thought to possess a certain immunity and the negro and Irish a certain liability thereto. The fact is, as shown by Dr. T. S. Sachs, consumption is very prevalent among the Jews of Chicago, and is so because of their living in unsanitary, bacilli-infected tenements by night and working by day in equally pestilential shops. To a limited extent they are less liable than some other nationalities on account of their better habits as regards vice and drink and the enforcement by their religion of better methods of preparing food, but they possess no natural immunity.

The reason consumption plays such appalling havoc among the colored and the Irish is to be found in their indifference to the laws of health, in their vices, as respects the negro, and in abuse of alcohol among the Irish. Mr. Ernest Poole is authority for the statement that of the tenement dwellers in New York City the Irish furnish the greatest number of victims to tuberculosis, and, in his opinion, because of their constitutions being undermined by drink and kindred excesses. In Chicago the Bohemians are said to be strikingly subject to pulmonary tuberculosis, not because of their nationality, but because of their aversion to fresh air in their homes. These are neat and clean as a rule, but the air in them is foul. The prevalence of phthisis depends on social conditions, not on nationality.

Tuberculous Food.—The consideration of the ætiology of pulmonary tuberculosis is not complete without the discussion of the importance to be attached to tuberculous food, and in particular to milk. As has been stated already, there is a divergence of opinion regarding the danger to be ascribed to milk from tuberculous cows. By von Behring and his adherents it is believed that the intestinal, glandular and bone tuberculosis seen in children is attributable to their ingestion of contaminated milk, and in this connection it may be stated that since the establishment of a governmental control of the dairy herds of Great Britain with a view to lessening tuberculosis among cattle there has been reported an appreciable diminution in the death rate among infants from tabes mesenterica.

The danger arising from the use of milk concerns mainly bottle-fed infants for the reason assigned by Behring, that their gastro-intestinal mucosa is protected only by an imperfect epithelial covering in the first few months of life. Yet, by many, it is believed that tuberculous milk and meat are liable to produce disease during the entire period of childhood if not at all ages.

By the opponents of this view it is held that the milk from cows is n in reality responsible for the intestinal or gland disease seen in children, or, more exactly speaking, that tubercle bacilli not of bovine origin create the disease—that germs derived from human beings have in some way been introduced either along with the food or independently thereof. For instance it is on record that several babies who were fed cow's milk by a certain nurse all contracted tuberculosis and died. It was supposed that the milk was tuberculous, but it has been proven that the milk became infected from the woman herself who had the habit of cooling each spoonful of milk by blowing upon it before feeding it to the infant, and as she had tuberculosis of a maxillary sinus she thus contaminated the milk.

The foregoing is narrated only to show how conflicting is the testimony concerning the danger from uncooked milk and flesh. Koch may be right in his opinion that bovine tuberculosis is not communicable to human beings, and yet I venture to assert that none of us would like to feed milk to our children or to drink it ourselves, which we knew came from a cow with tuberculous ulceration of her udder. Therefore let us physicians continue to impress upon our patients and communities the wisdom of all measures calculated to eradicate this disease from our cattle.

The relationship existing between primary intestinal or gland tuberculosis and consumption lies in disease of the bronchial lymph nodes to which the pulmonary tuberculosis of children is secondary; that is, if a tuberculous focus exists anywhere in the body, there is always the possibility of its breaking into the circulation and thus setting up miliary tuberculosis of the lungs or general system. From every standpoint, therefore, it is the part of wisdom to enforce all measures that may guard against infection by tubercle bacilli from whatever source.

Summary.—Pulmonary tuberculosis is a specific infective disease. The infective agent is the tubercle bacillus. This organism is widely distributed and finds ready access to the lungs by inhalation, by the blood and by the lymph channels. Whatever lowers tissue resistance on the part of the individual renders him susceptible to the action of the germ. Social conditions that keep alive the bacillus in the dust or atmosphere or favor its dissemination through the community are to be regarded as ætiological factors.

Finally, the so-called tuberculous diathesis or inherited predisposition to consumption is a belief based on wrong hypotheses. The whole subject must be looked at from the viewpoint of infection and not hereditary tendency.

Morbid Anatomy. *The Tubercle.*—The lodgment of the bacillus in a living tissue is followed by its rapid multiplication and growth. Consequent thereon is proliferation of the cellular elements of the invaded tissue, i. e., of the endothelial and connective-tissue cells together with the emigration and collection of leucocytes around this point. Thus is produced a small nodule known as *tubercle* which may vary in size from that of a millet seed to that of a small pea. To such tiny masses the term miliary tubercles is applied. They may present a grayish-white translucent appearance or they may, when caseous, have a yellowish color.

Examined microscopically (Plate VII) these nodular tubercles, as they are called to distinguish them from uniform tuberculous infiltration, are seen to be composed of three kinds of cells: (1) Epithelioid cells which are spheroidal or polyhedral cells having a clear nucleus and often containing the bacilli. (2) Giant cells which are large multinuclear cells formed either by increase in the nucleus and protoplasm of individual cells or by coalescence of several cells. These giant cells, once thought characteristic of tubercle, exist in varying number and appear to be in inverse ratio to the number of bacilli present in the nodule. (3) Leucocytes, at first of the polynuclear variety (phagocytes), but later on the lymphocytes which do not suffer such rapid destruction as do their predecessors.

After a time a fine stroma of fibrous tissue develops at the outer margin of the tubercle which is an attempt at encapsulation and arrest of the nodular growth. Blood-vessels are not seen in the tubercle. In the center, and in some instances throughout the tubercle, may be found a yellowish homogeneous material which represents cells that have undergone caseous degeneration, the ultimate fate of tuberculous tissue in most instances.

Infiltrated Tubercle. — In some cases the pulmonary tissue does not present individual nodules but is infiltrated or replaced by tuberculous tissue. When of a grayish-white appearance, still early in the process, it is spoken of as Laennec's "gray infiltration," but when, under the action of the bacillus, it has suffered coagulation necrosis, it

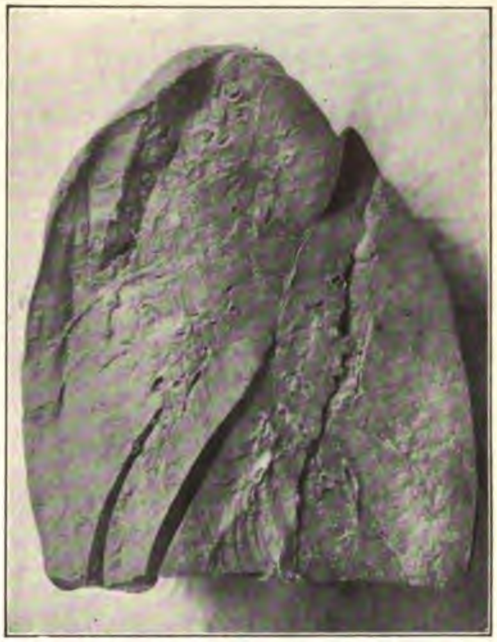


FIG. 45.—Pneumonic phthisis.

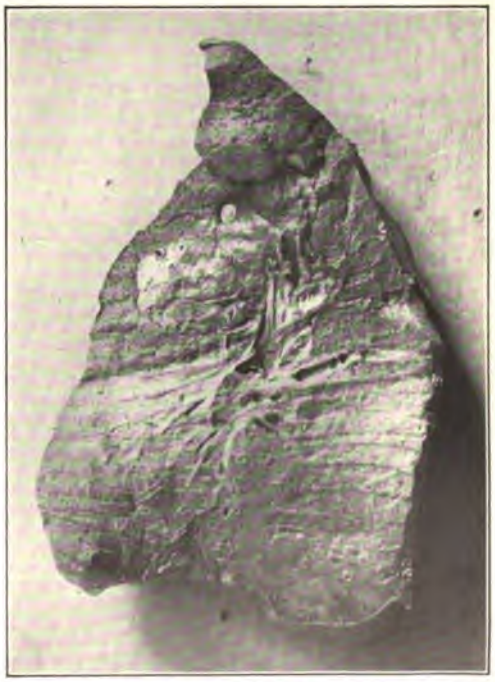


FIG. 46.—Pneumonic phthisis, involving one lobe.

constitutes the so-called "cheesy pneumonia" of English writers, or Laennec's *infiltration tuberculeuse jaune*.

Microscopically, these areas of tuberculous infiltration are seen to be made up of minute tubercles surrounded by zones of inflammation, the air-vesicles being filled with inflammatory exudation products and with proliferated and fatty alveolar epithelium. Such a tuberculous infiltration may invade limited areas or an entire lobe (Figs. 45 and 46) or even a whole lung. As these

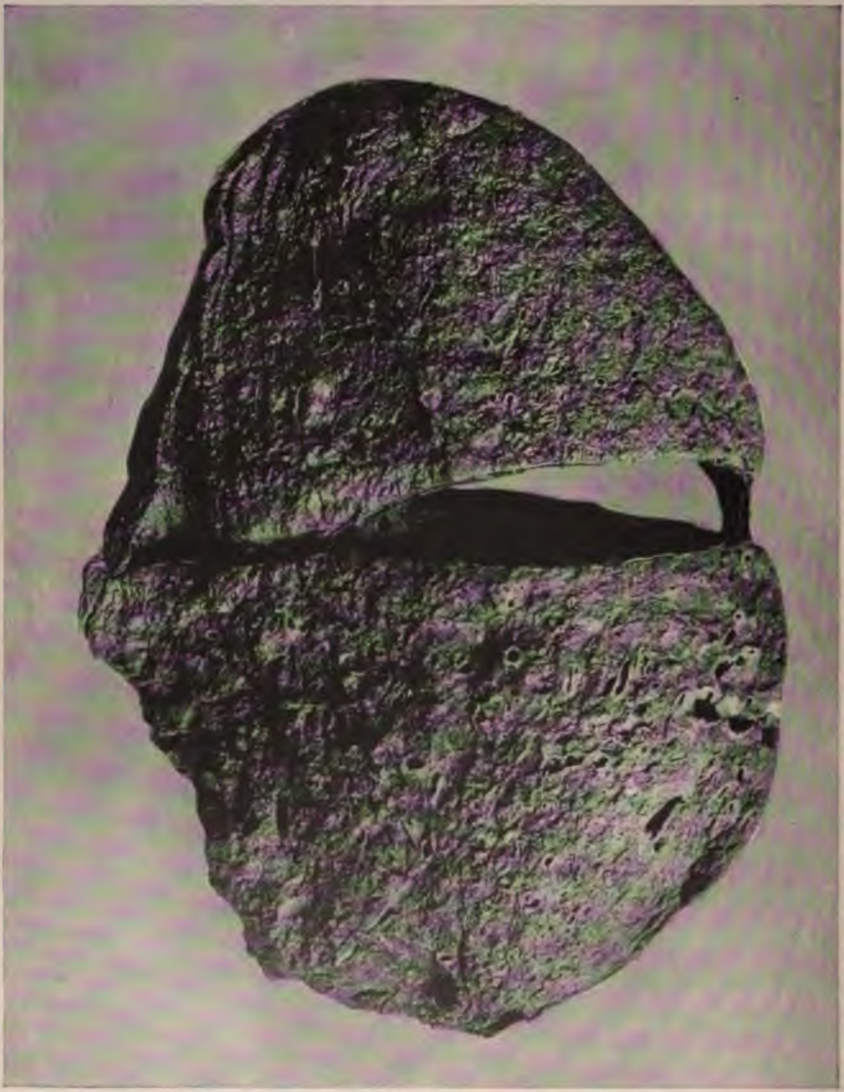


FIG. 47.—Acute miliary tuberculosis of lung. Note adhesion across interlobar fissure.

inflammatory products tend rapidly to undergo coagulation necrosis, these infiltrated zones soon become caseous and necrotic. According as this process goes on rapidly or slowly we distinguish acute or chronic tuberculous pneumonia.

Changes in the Lung Depending upon the Route of Infection.—The manner in which the bacilli gain access to the lung determines the anatomical picture, and very largely also the clinical manifestations. Thus, if it be *hæmatogenic,* that is, if, in consequence of the rupture of a tuberculous focus into a blood-vessel in some distant part of the body or in the lung itself, bacilli are carried by the blood-stream, they may lodge in the wall of a pulmonary capillary or in the interstitial connective tissue and there excite a productive inflammation. Consequently, miliary tubercles become scattered throughout the organ (Figs. 47 and 48). In such an event they form but a pulmonary manifestation of an acute general miliary tuberculosis.

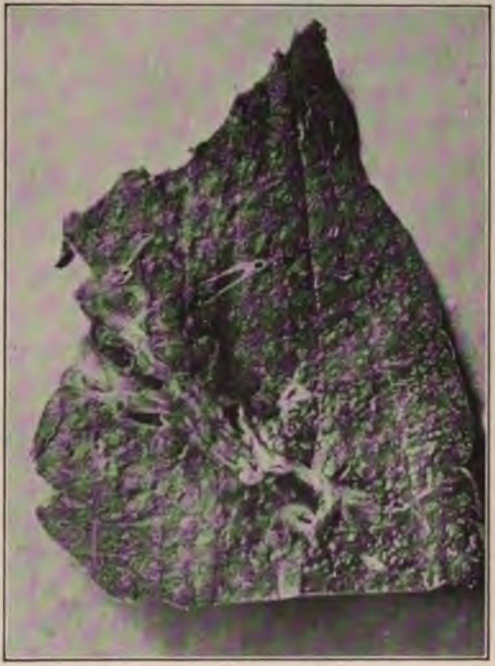


FIG. 48.—Miliary tuberculosis, less acute than that in Fig. 47.

In some instances the process seems to be limited to a proliferative inflammation when the lung is found thickly beset with minute grayish-white translucent tubercles. In other cases there is also a zone of exudative inflammation surrounding the tubercles and the alveoli are blocked with the inflammatory exudate, forming small areas of tuberculous broncho-pneumonia. The tubercle proper is the result of the action of the bacillus, while the areas of exudative pneumonia are thought due to the action of toxins created by the germ. The process may be very rapid, when it is spoken of as acute, or it may be slow, and then forms the chronic miliary tuberculosis.

In the *lymphogenic form,* which is seldom encountered and by some writers is not mentioned at all or is considered exceedingly doubtful, the tuberculous process attacks the lung secondarily to disease of the pleura or some of the adjacent organs, as vertebræ, ribs, mediastinal or bronchial glands. In the case of the glands they become adherent to the pleura and thence the tubercles spread radially in all directions (Fraenkel).

The most usual mode of origin of pulmonary tuberculosis is that known as *aerogenic,* or by inhalation. The bacilli may be inhaled along with dust constitutes the so-called "cheesy pneumonia" of English writers, or Laennec's *infiltration tuberculeuse jaune*.

Microscopically, these areas of tuberculous infiltration are seen to be made up of minute tubercles surrounded by zones of inflammation, the air-vesicles being filled with inflammatory exudation products and with proliferated and fatty alveolar epithelium. Such a tuberculous infiltration may invade limited areas or an entire lobe (Figs. 45 and 46) or even a whole lung. As these

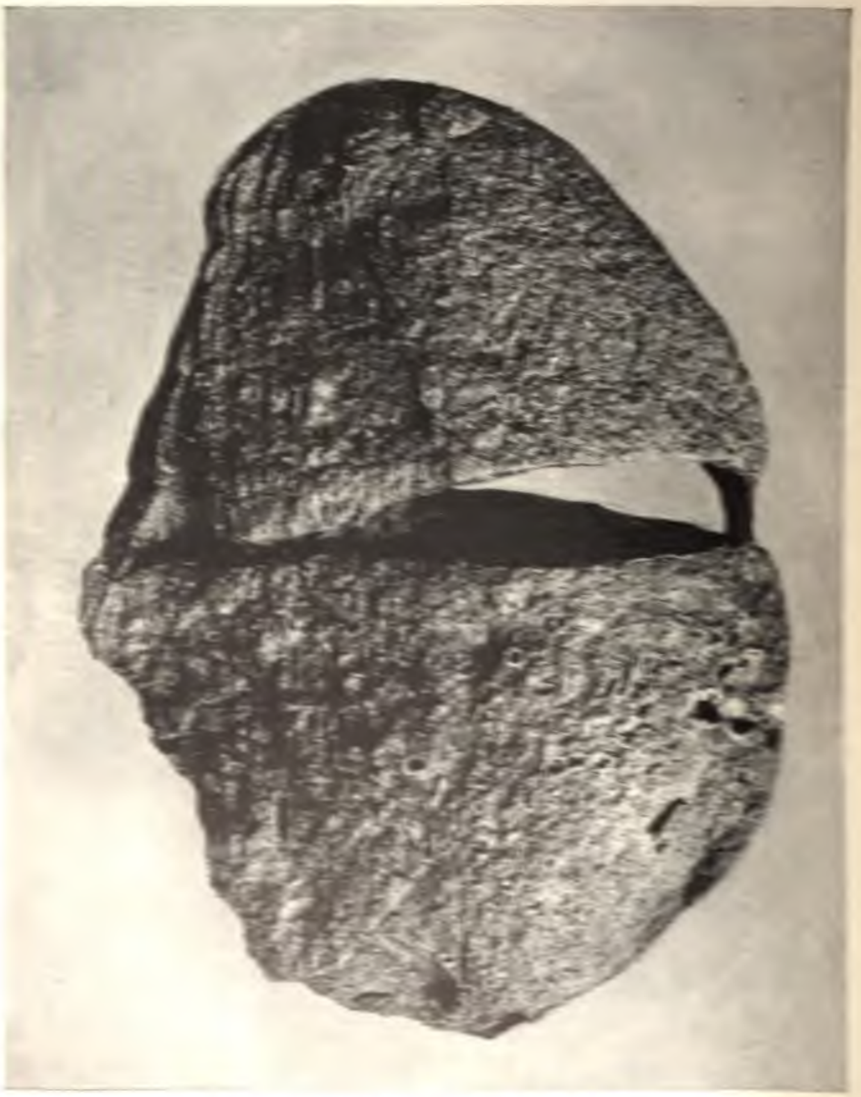
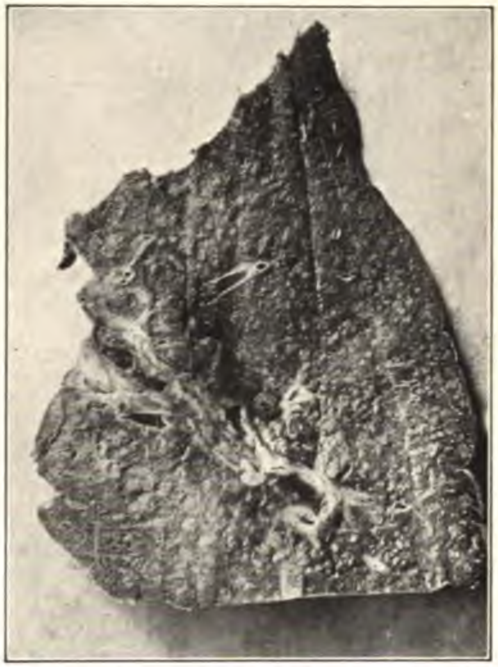


Fig. 47.—Acute miliary tuberculosis of lung. Note adhesion across interlobar fissure.

inflammatory products tend rapidly to undergo coagulation necrosis, these infiltrated zones soon become caseous and necrotic. According as this process goes on rapidly or slowly we distinguish acute or chronic tuberculous pneumonia.

Changes in the Lung Depending upon the Route of Infection.—The manner in which the bacilli gain access to the lung determines the anatomical picture, and very largely also the clinical manifestations. Thus, if it be *hæmatogenic*, that is, if, in consequence of the rupture of a tuberculous focus into a blood-vessel in some distant part of the body or in the lung itself, bacilli are carried by the blood-stream, they may lodge in the wall of a pulmonary capillary or in the interstitial connective tissue and there excite a productive inflammation. Consequently, miliary tubercles become scattered throughout the organ (Figs. 47 and 48). In such an event they form but a pulmonary manifestation of an acute general miliary tuberculosis.



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The most usual mode of origin of pulmonary tuberculosis is that known as *aerogenic,* or by inhalation. The bacilli may be inhaled along with dust or may be aspired in consequence of rupture and discharge into a bronchus of a caseous node or focus of old disease at the apex. The results are essentially the same no matter how many bacilli are thus carried into the lung, but the extent of the mischief is determined by their number.

Acute Tuberculous Broncho-pneumonia.—In this form, which generally owes its origin to the inhalation of bacilli, the process is characterized by the development of an exudative inflammation which, although associated with miliary nodules, greatly preponderates over these. Such a tuberculous pneumonia may be met with in either of two forms, namely, as circumscribed zones of broncho-pneumonia or as a diffuse process (Fig. 49). The former is frequently encountered in children and is then apt to be mistaken for chronic broncho-pneumonia, while the diffuse form, since it generally occurs at the apex and may involve the whole or greater portion of the lobe, is quite likely to be regarded as an ordinary croupous pneumonia.

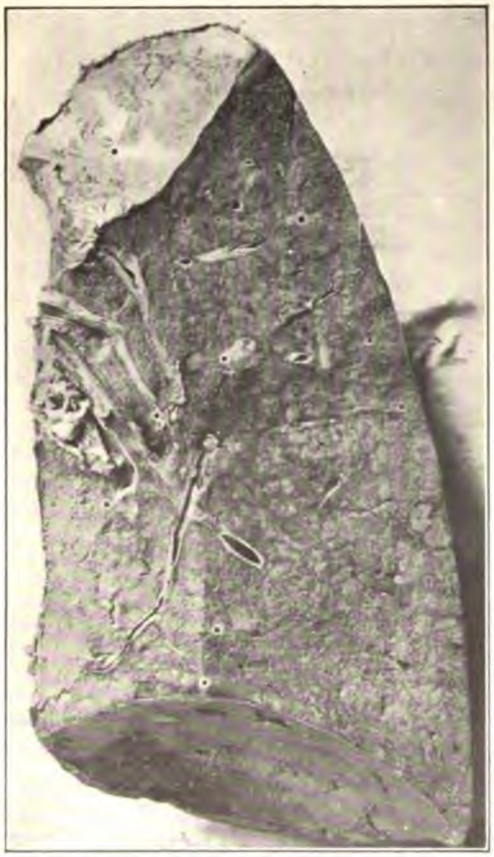


FIG. 49.—Tuberculous broncho-pneumonia.

The weight of the lung is increased and if the whole organ is consolidated it may even weigh twice as much as its fellow. It is of a grayish-white color which may be distinguishable through the pleura while, on section, reddish zones or bands may here and there be seen corresponding to areas of congestion. The cut surface is smooth or in places slightly granular but the consistency of the lobe is less firm than in ordinary pneumonia.

Along the inferior margins the lobe may still crepitate but for the most part the consolidated tissue is completely airless. In some instances distinct tubercular nodules may be visible in immediate proximity to an old vomica or cicatrix in the apex, but the microscope shows that, for the most part, the consolidation is due to a compact cellular infiltration, the desquamative pneumonia of Buhl.

Cavities are very commonly present, varying much in size and number. There may be numerous small ones or but a single large one with ragged necrotic walls (Fig. 50). In man this softening and excavation seems, from Prudden's experiments, due to the admixture of other germs, generally streptococci. As this necrosis takes place rapidly and spares only the blood-vessels the cavity is apt to be traversed by the vessels either unsupported and intact or not infrequently displaying a loss of continuity at some point in consequence of the walls having been attacked by the ulcerative process. It is such a perforation in the vascular coats which is responsible for the profuse hæmorrhages so often seen in acute pneumonic tuberculosis. In such a case the cavity is likely to contain blood.

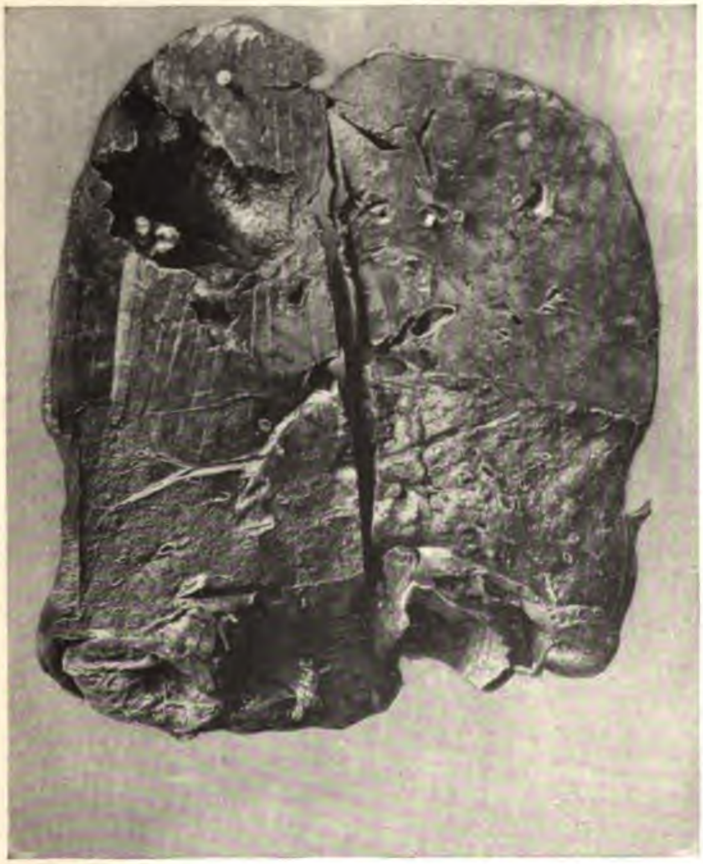


FIG. 50.—Tuberculous broncho-pneumonia with cavity formation.

The large bronchi may show signs of inflammation and the opposite lung may contain a few tubercles. In less rapidly fatal cases the lung primarily affected may show an attempt at arrest of the destructive process, while in the opposite lung the upper lobe displays the changes of recent pneumonia with or without necrosis and excavation. In other instances the superior portion of the lung may contain old, partially healed lesions and the lower lobe be the seat of fresh pneumonic infiltration, evidently the result of recent infection from the original focus at the apex.

The form of acute tuberculous pneumonia just described constitutes what is variously termed Galloping or Quick Consumption, Phthisis Florida, Acute Phthisis, Acute Pneumonic Phthisis, Desquamative Pneumonia (Buhl), and, according to the extent and rapidity of the destructive process, its duration may vary from two or three weeks to several months. I recall a case in which the right lung was first affected and went on to rapid excavation yet without hæmorrhage. The left one then showed signs of extensive involvement and softening, and the process terminated fatally in six weeks.

Chronic Pulmonary Tuberculosis.—In this form, the one usually encountered in ambulatory patients, there is a striking variety in the appearances. Some of these represent an attempt at arrest and repair while others exhibit the destructive nature of the process, and still others the tendency to active extension of the tuberculous affection into fresh parts. The initial lesion is generally in one apex whence it progresses more or less slowly downward, invading the apex of the lower lobe before it reaches the base of the upper in front. The opposite lung also becomes involved soon or late in the process, and hence, on *post-mortem* examination, both lungs display changes of a similar nature but of very different extent.

Primary Seat of the Tuberculous Lesion.—As originally taught by Laennec the initial site of tubercles in the lung is at the apex of the upper lobe. In a small number of instances (1 in 500, Percy Kidd) the lower lobe may be the first involved. The importance of a precise knowledge of the seat of predilection is great and has a practical bearing on its early clinical detection. Hence I will here state the results of Kingston Fowler's researches on this point.

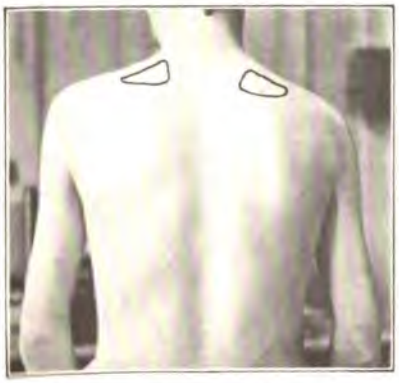


FIG. 51.—Location of usual primary focus of tuberculosis.

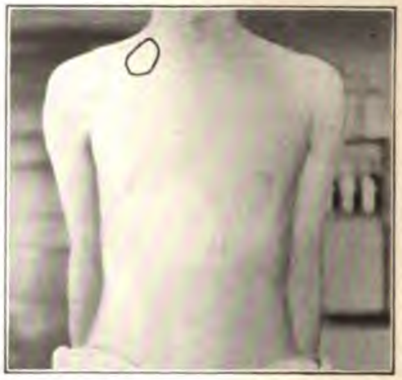


FIG. 52.—Anterior point at which signs appear from primary focus.

According to this author the primary tuberculous focus is, in the over-ming majority of cases, at a point about an inch or an inch and a half below the extreme summit of the lung and somewhat nearer its posterior and outer border. From this point it spreads downward and backward and hence should be sought for in the suprascapular region (see Fig. 51), since clinical signs of disease may be discovered here before it appears in front. "From this primary focus," says Fowler, "which in front corresponds either to the supraclavicular fossa or to a spot immediately below the center of the clavicle (Fig. 52), the lesions often spread downward along the anterior aspect of the upper lobe, about three fourths of an inch within its margin, frequently occurring in scattered nodules separated, perhaps, by an inch or more of healthy tissue. It is not unusual to find in these scattered nodules the only evidence of disease on the anterior aspect of the lung, when posteriorly excavation has advanced to such a degree that but little more than the pleura remains."

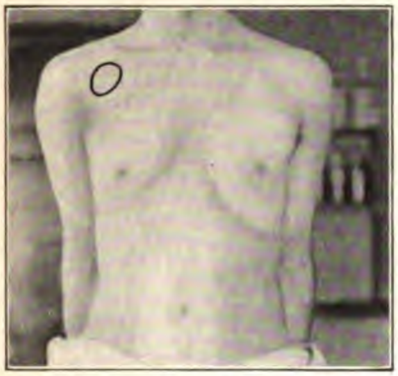


FIG. 53.—Less frequent site of primary focus of disease.

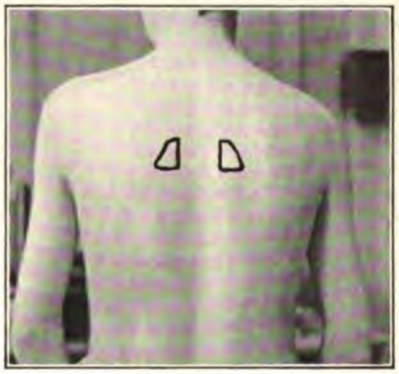


FIG. 54.—Site of secondary lesion at apex of upper lobe.

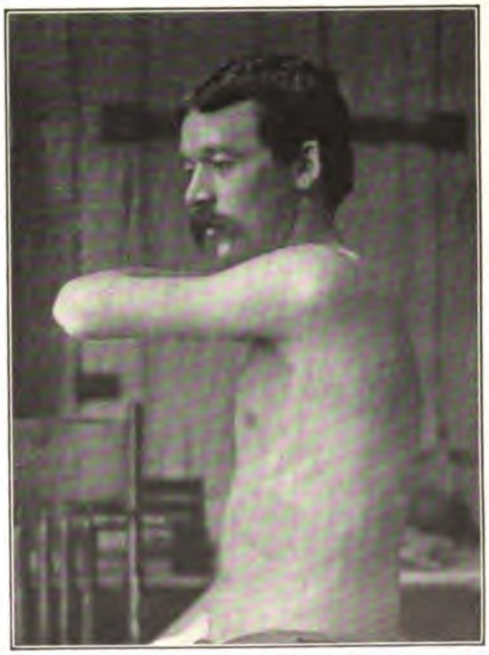


FIG. 55.—Position of arm in which vertebral border of scapula defines interlobar fissure.

A second but less usual situation of the primary focus is shown in Fig. 53, and corresponds to the first and second interspaces below the outer third of the clavicle. From this latter initial site the lines of extension are also downward, so that clinical evidence of the disease is found in an oval-shaped area at the outer rather than the inner part of the upper lobe.

The lower lobe is very rarely, if ever, the seat of a primary lesion but shows advanced changes secondary to those of the upper lobe and long subsequent to the destructive process behind in the lower lobe.

The remarkable predilection of the apex of the upper lobe to tubercle has been variously explained. It has been attributed to relative anæmia of this portion of the lung, to defective ventilation and, by Birch-Hirschfeld, to the anatomical peculiarity of its air-tubes which favors the stagnation of the respired air at certain points and hence the deposit of bacilli.

According to this late distinguished pathologist the bronchus leading to the upper lobe ascends in a nearly vertical direction, so that the column of air moving within it passes in a direction opposite to that of the air in the trachea. For this reason the movement of air within the apical bronchus is enfeebled, while in the small tube leading to the superior and posterior portion of the apex there is a tendency to stagnation of the air at any point where it is met by a stronger column of air passing at a right angle across the mouth of the bronchiole.

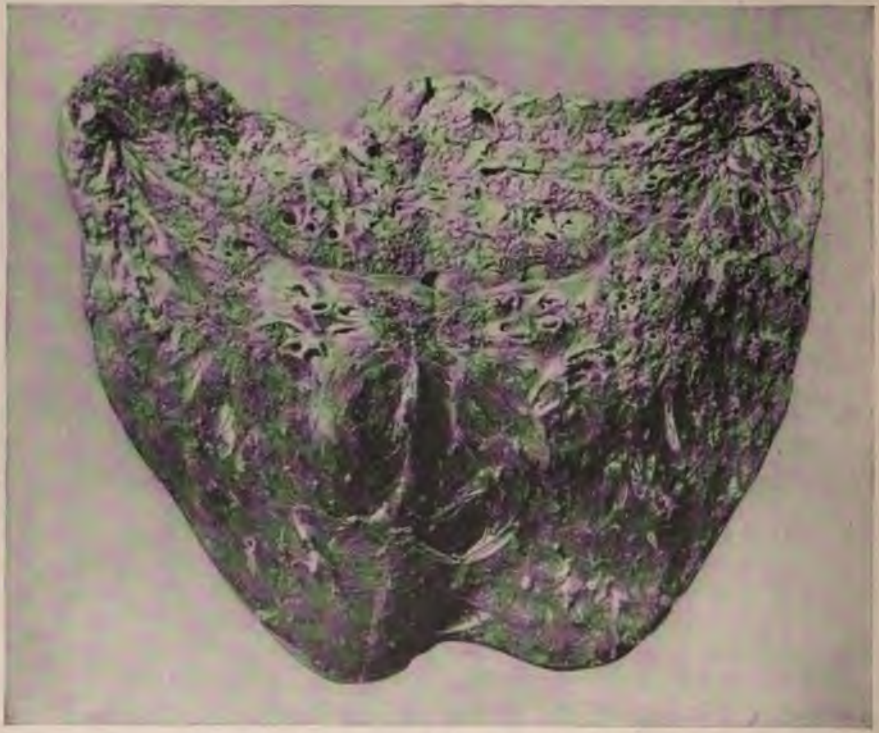
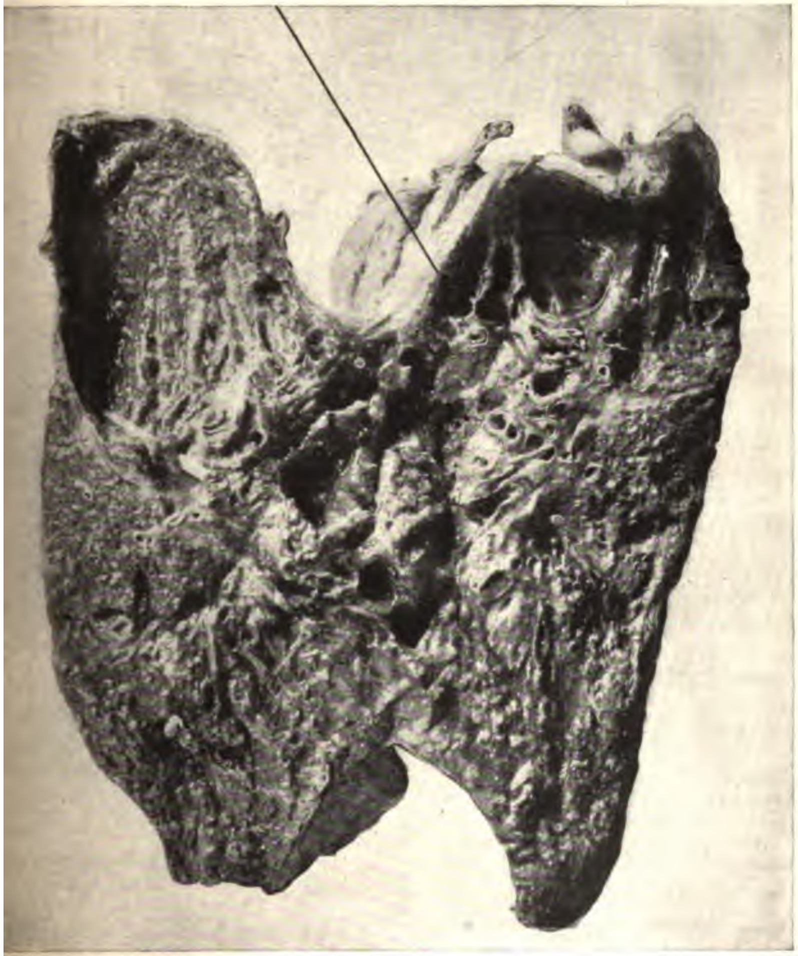


FIG. 56.—Fibro-caseous tuberculosis of right apex.

With such an anatomical peculiarity predisposing to air-stagnation, it only ires some additional factor to determine disease. Such an added element be found in a local catarrh or in defective development of the tube which en in poorly built chests.

n young children this peculiar anatomical arrangement of the apical chus does not exist, according to Birch-Hirschfeld, and, moreover, the



57.—Caseation and cavity formation on large scale. Left lung of same case as Fig. 56. ine indicates cut end of a thrombosed vessel. Wooden probe passes through bronchial pening into cavity.

ter elasticity and mobility of the thorax maintains a better ventilation of upper lobe than exists later in life. Hence, one of the reasons why, prior the fifth year, pulmonary tuberculosis finds its primary seat not at the x but in the middle and inferior parts of the lung. Other reasons will found set forth in the description of tuberculosis in childhood.

If Birch-Hirschfeld's statements are correct, they serve to make clear the injurious influence of certain occupations as respects the development of tuberculosis. It has long been known that workers at a bench, as jewelers, tailors, shoemakers, etc., are particularly prone to acquire consumption. Not only are such workers exposed to the inhalation of contaminated dust, but in consequence of their sitting attitude and the nature of their work their heads are bent forward, their chests are cramped and their respiration is enfeebled.

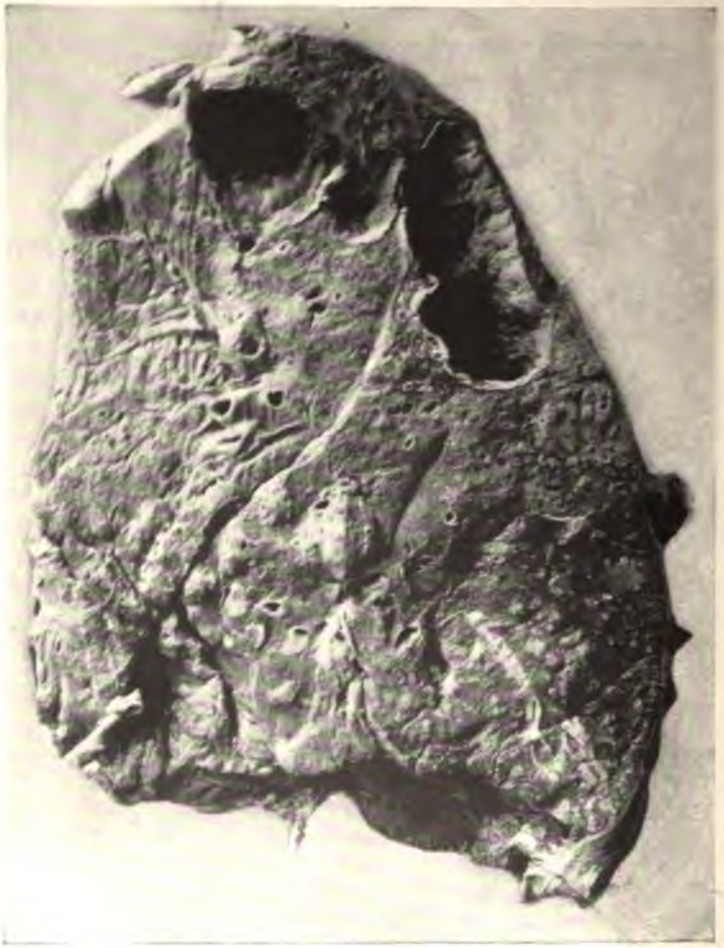


FIG. 58.—Cavities with well-defined walls.

The ingress and egress of air at the apices of the lungs is hindered and the natural anatomical weakness is intensified. Under such occupational conditions, therefore, the retention and deposit of bacilli at the apex of the lung is furthered and we can understand why bench-workers, and in particular workers in sweatshops, form so large a contingent of consumptives.

The site of the secondary lesion in the lower lobe is about an inch or an inch and a half below its highest part, at a point on the chest-wall (see Fig. 54) about midway between the fifth dorsal spine and the vertebral border of the scapula. This region is commonly infiltrated early, at a time before the opposite lung is involved and before the base of the upper lobe has been reached in front. The line of extension in the lower lobe is along the interlobar sæptum, which line on the chest-wall corresponds roughly to the inner border of the scapula when the hand grasps the shoulder of the opposite side and the arm is elevated so as to bring the elbow on a plane above that of the shoulder (see Fig. 55). The area of extension thus uncovered for the purpose of chest-examination is wedge-shaped and should be carefully sought for when signs at the apex are obscure.

Description of the Lesions.—As previously stated these are varied both in appearance and extent. There are usually tubercular nodules scattered through the lung, especially in the lower lobe in chronic miliary tuberculosis, or clustered radially about an old cavity or a partially calcified caseous mass at the apex. The microscope may also reveal areas of tuberculous infiltration which, to the naked eye, appear as grayish-white zones.

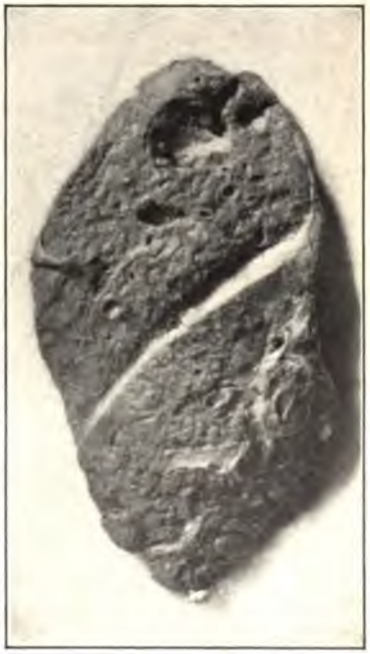


FIG. 59.—Small cavities.

Yellowish areas of caseation are also scattered through the lung, in some cases intimately associated with fibrous tissue (Fig. 56), in others with but little fibroid tissue and abundant cavity formation (Fig. 57). The amount and preponderance of cheesy degeneration seems, therefore, to stand in direct relationship to the destructive nature of the process. Hence, in very chronic cases caseation is less pronounced a feature than is the tendency to arrest.

Fibrous tissue is one of the means resorted to by nature for the arrest of the tuberculous process, and hence in some cases the lungs display an unusual proportion of this tissue (fibroid phthisis). In all, however, there is more or less formation of dense fibrous tissue encapsulating or connecting caseous and tuberculous zones and especially abundant in the neighborhood of old cavities.

Zones of broncho-pneumonia are also seen surrounding terminal bronchioles which, together with the alveoli, are filled with exudative and epithelioid cells. If one of these areas of peribronchial tuberculosis be divided transversely the end of the cut air-tube appears as a minute opening in the midst of a mass of cells, and the microscope may show minute miliary tubercles scattered in the walls of the terminal bronchioles. Such broncho-pneumonic zones in time become caseous and necrotic and form the seats of future vomicæ when life lasts a sufficient length of time, or, escaping such actively destructive process, they may become encapsulated by fibrous tissue.

Cavities usually constitute a striking feature of the lungs in chronic phthisis (Figs. 58, 59, 60, 61). They may vary greatly in number and size as well as display all possible differences in the necrotic process. There may be but a single large vomica occupying the major portion of the upper lobe, or several may unite in such a manner as to form what is known as an anfractuous cavity. If the changes have taken place slowly and the tendency to fibrosis is pronounced, the cavity may be encapsulated, its walls be comparatively smooth and the vomica be empty—the so-called *dry cavity.*

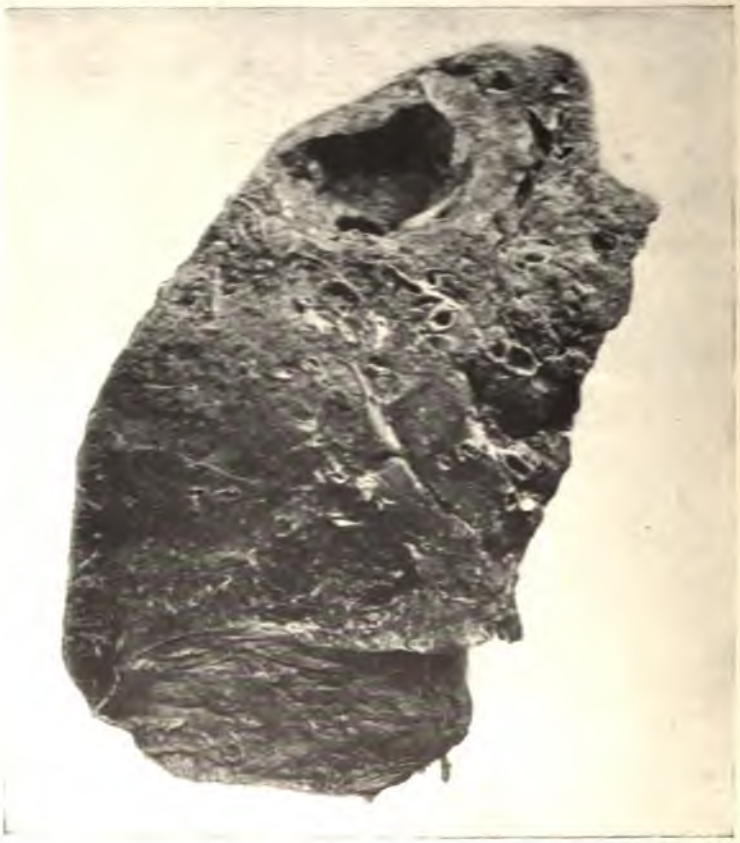


FIG. 60.—Large single cavity.

Not unfrequently the cavity is lined with granulation tissue from which pus is exuded. In other instances the cavity is surrounded by caseating material so that its walls are necrotic and ragged. In such an event it is apt to contain more or less necrotized tissue, pus and blood, and if its walls are gangrenous it may emit a horribly offensive odor.

Either projecting from its sides or traversing its interior may be seen blood-vessels shriveled into fibrous cords and impervious, or still serving as channels for the blood-stream (Fig. 57). In the latter event the vessel-coats are apt to present larger or smaller dilatations (*pulmonary aneurysms*). It is such aneurysmal bulgings which, by their rupture, occasion the profuse and even fatal hæmoptyses seen in chronic ulcerative phthisis. The divided end of a bronchus may be discovered in the wall of the cavity (Fig. 57) and, according to West, it is sometimes remarkable how minute such an opening into the air-tube may appear.

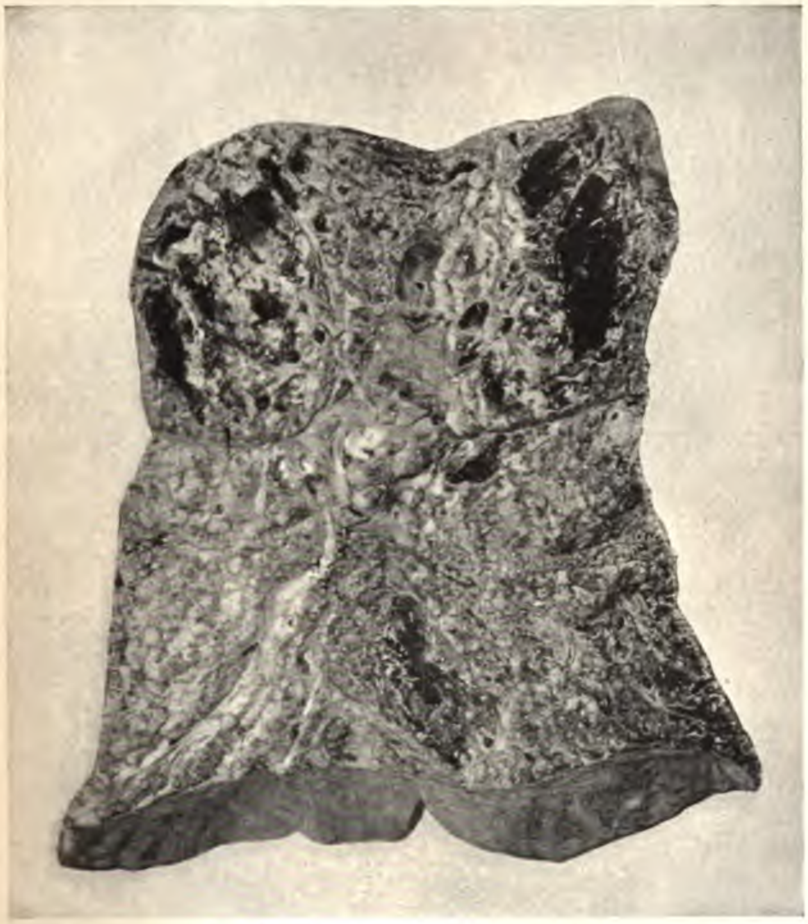


FIG. 61.—Ragged cavity, forming rapidly.

The cavities of chronic tuberculosis present also a remarkable variety in

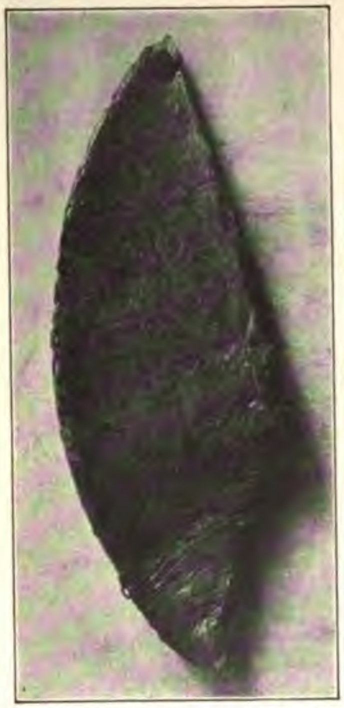


FIG. 62.—Miliary tubercles on the pleura.

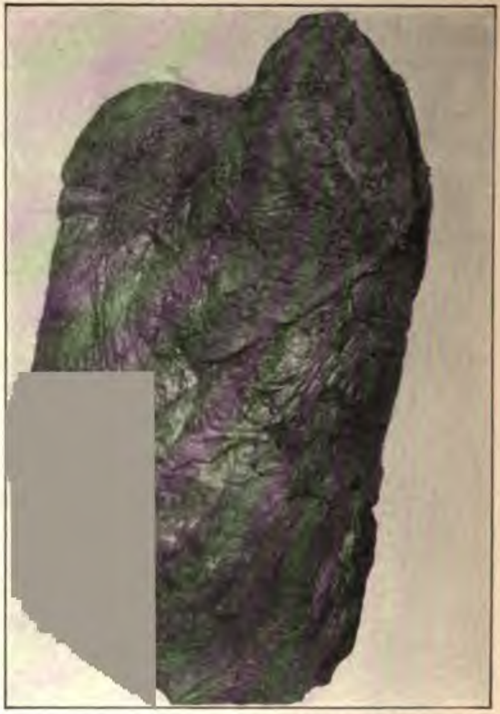


FIG. 63.—Small puckered scar on pleura near apex.

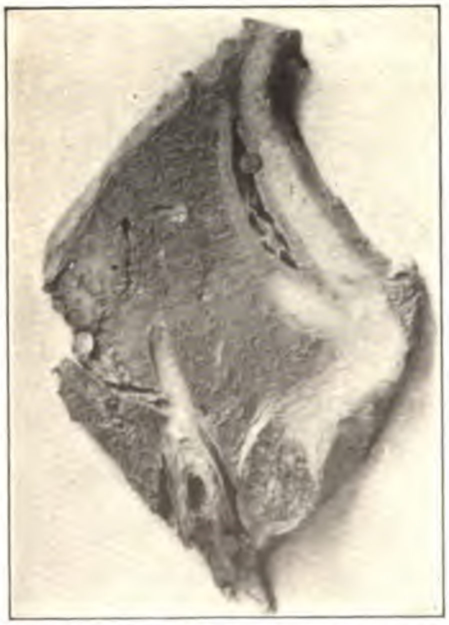


FIG. 64.—Pleura thickened and caseous with tuberculous lymph gland.

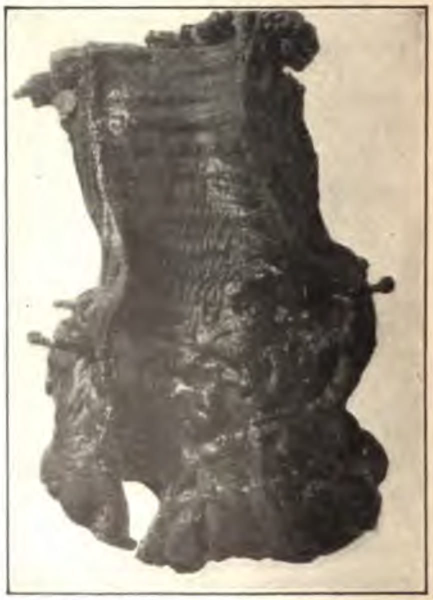


FIG. 65.—Tuberculous ulceration of trachea.

shape. They may be fairly round and regular or they may display deep indentations which give to their periphery an irregular outline like the broken coastline of some of our inland lakes. It is not uncommon for a vomica to be long and narrow so that, beginning near the apex of the lung, it extends deeply downward or downward and backward into the lower lobe. According to Fowler, it is not very unusual for the individual areas of tubercle distributed along the anterior margin of the upper lobe in front to break down and coalesce so as to form a slender vomica extending deeply toward the base.

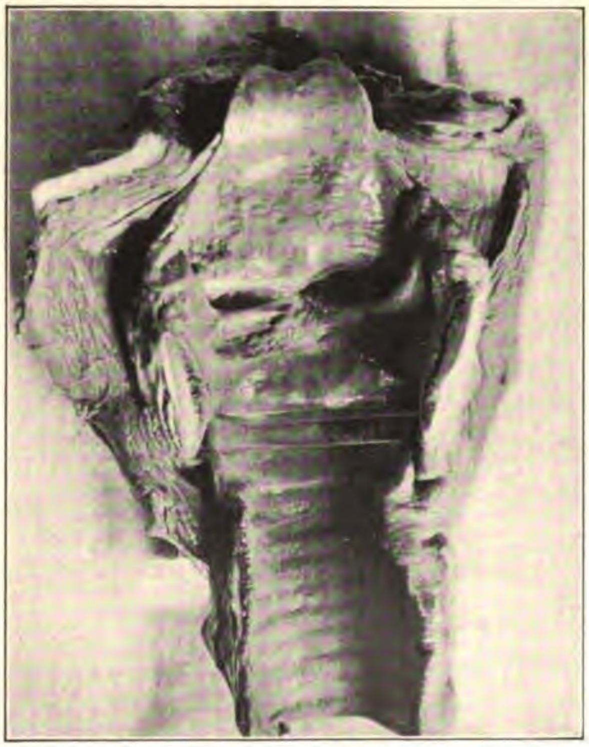


FIG. 66.—Tuberculous ulceration of larynx.

In some instances it is extremely difficult to decide whether a cavity is a true vomica or is the ulcerated and dilated bronchus.

I recall the case of a young man whose heart was displaced to the right in consequence of firm old adhesions, and whose entire left lung had been ulcerated away, so as to form a huge cavity, its only wall being the thickened pleura lined throughout with cheesy material. Perhaps an eighth of an inch thick at the apex this lining of caseous matter was from a quarter to half an inch in thickness at the base. The sealed main bronchus opened into it above and its floor was covered with odorless creamy pus. It was therefore a huge pyopneumothorax. The right lung showed extensive old lesions with so little healthy parenchyma remaining that it was difficult to comprehend how the young man had breathed at all. Numerous gray granulations were distributed in the pericardium.

The pleura overlying the lung is the seat of dense fibrous adhesions, or it may be firmly united to the parietal pleural membrane throughout. Nodular tubercles may be seen here and there on the pleural surface (Figs. 62, 63, 64). It is not very uncommon to find that a superficially situated vomica has ruptured into the pleural sac and set up a pyopneumothorax.

Associated Lesions.—The bronchial lymph glands at the root of the lungs are very commonly tuberculous or caseous (Fig. 64). The mesenteric and retroperitoneal lymph nodes are also likely to be the seat of tubercle, and miliary nodules may be scattered throughout the various abdominal viscera.

A very frequent accompaniment of pulmonary tuberculosis is infiltration and ulceration of the larynx and vocal cords (Figs. 65, 66). There may also be tubercles scattered through the pericardium or, very rarely, in the heart-walls. Meningeal tuberculosis is likewise found in some instances, especially in children. Such a wide distribution of tubercular nodules is only likely to be discovered in cases that, after pursuing a chronic course, have terminated in general miliary tuberculosis.

CHAPTER XXV

PULMONARY TUBERCULOSIS—*Continued*

Symptoms. Chronic Pulmonary Tuberculosis.—In the great majority of cases of pulmonary tuberculosis the disease manifests its presence so insidiously that the patients do not take to bed but perform their daily tasks long after the time when they should have sought medical aid. The cases are ambulatory and essentially chronic. For this reason it seems best to present the symptomatology of pulmonary tuberculosis as nearly as possible in the way it is ordinarily observed. Before beginning its description, however, it will be well to spend a little time in discussing the various stages into which it is customary to divide the complaint.

Incipient Stage.—This, as the term signifies, is the stage or period of development and is therefore the earliest or first stage. Properly it includes the period when tubercles are forming in the lungs before they have occasioned obvious constitutional disturbance, i. e., the time of productive inflammation. It embraces also the period when general and perhaps local or pulmonary symptoms attract attention, that is, the time of localized bronchial catarrh, and it merges gradually into what is known as the second stage.

This first or incipient stage is progressive and presents differences in the intensity of its manifestations at different periods in its progress. Accordingly, attempts have been made to subdivide this stage, and various terms have been suggested for different periods of it. Thus, Bayle, who was Laennec's teacher, applied the term Phthisis Occulta to the period of the disease when the lesion is too circumscribed to give rise to recognizable local signs. More recently a French writer, Granger, has adopted Bayle's classification under the name *Periode de Germination.*

By English and American writers this is spoken of as the *latent stage.* It is meant to embrace the time when tubercles are forming and declaring their presence by indistinct constitutional symptoms, though they cannot be ordinarily detected by physical examination of the lungs. The diagnosis at this time must be either inferential or based on general considerations. Bacilli are not discoverable at this stage and hence it is sometimes spoken of as the *prebacilliary stage.*

This term is somewhat less restrictive, however, inasmuch as it may include that time when very slight, though recognizable, local signs may be present, yet without sputum or, at all events, sputum containing tubercle bacilli. When this stage has progressed to the production of scanty mucous expectoration it has reached the *catarrhal stage,* as some choose to designate it.

A moment's thought will convince one, I think, of the difficulties in the way of so exact a subdivision of this first stage, and therefore, in my opinion, it is more practicable to adopt the term *incipient,* although open to the objection that after the pulmonary lesion has once formed it is no longer in a formative stage.

Stage of Softening.—In this period of the disease, sometimes spoken of as the *second stage,* both constitutional and pulmonary symptoms have become too obvious to be overlooked. The lesion is no longer formative, or, if productive inflammation is still going on secondarily, it is disguised by the evidence of caseation and softening. In addition to tubercle bacilli other micro-organisms are discoverable in the sputa, chiefly streptococci, but sometimes staphylococci and the bacillus pyocyaneus.

It is therefore a stage or condition of *mixed infection* and the disease has reached a time when it can be properly spoken of as *consumption.* Indeed, some clinicians restrict the term consumption to that period in its course when the disease is no longer a tuberculosis pure and simple but is one of mixed infection. It embraces therefore the so-called second and third stages.

In a certain sense, this period of softening may be termed a second stage since it really is the manifestation of a second process, the process to which tubercle is always liable and to which, in most instances, it eventually succumbs. It is no longer an early stage, if by this is meant the period of tubercle formation, and yet, as measured by lapse of time, it may be early in the course of the pulmonary complaint. However, since caseation and softening mark a relatively late condition in the life-history of tubercle, this stage is plainly distinguishable from the stage of incipiency, and this latter alone should be designated as early. This latter merges insensibly into the stage of softening, and with the understanding that a sharp dividing line cannot be drawn I choose to adopt the term stage of softening in preference to second stage. For sake of convenience, however, I shall use interchangeably both second stage and stage of softening.

Stage of Excavation.—This term, which explains itself, embraces that period in the course of the disease when the softened caseous material is being expectorated and cavity is forming. It follows the second stage so gradually that a distinct demarkation between the two is impossible. It is the last or terminal stage in most cases, and hence is often so designated while, in contradistinction to the incipiency, it is characterized as the *late stage.*

All these stages are based on clinical or symptomatic considerations, not on anatomical basis, since tubercle formation, caseation and excavation may all and frequently be found conjoined, though in varying proportions. That these terms are open to objection is recognized, but that no others can be devised which are more satisfactory is proven by the fact that they have been the basis of classification ever since the time of Laennec.

General Clinical Picture.—As will be shown later on, the disease may set in variously, but in the great majority of cases the first symptom to attract attention is cough. There is a period during which the individual has a slight, dry hacking cough to which he either pays no attention or which he attributes to bronchial irritation. Suddenly this symptom becomes decidedly troublesome and he thinks he has taken cold. In some instances medical aid is sought, but many times recourse is had to some cough mixture obtained at a drug store.

If the remedy contain some preparation of opium, as is generally the case, the cough is ameliorated for a time. As a rule, however, the fancied bronchitis persists or increases in severity, appetite and strength fail somewhat and a physician is consulted. Frequently the signs of apex mischief are so insignificant as to be overlooked, or the examination is superficial and the physician confirms the patient's notion by the statement that the condition is only a bronchitis.

In reality there is likely to be slight dullness circumscribed and confined to one or the other suprascapular fossa, with bronchial breathing in this area and possibly fine crackling râles heard at the end of inspiration. In other instances the percussion-note seems unchanged or is slightly tympanitic and the breath-sounds are only broncho-vesicular, rough, as they are called. Such changes are not conspicuous and either on this account or because the examination is restricted to the front of the chest they are not detected.

If the case be pronounced a bronchitis, another cough mixture is prescribed and the patient is dismissed with the assurance that the cough will soon disappear. The comforting prediction is not fulfilled, however; the cough remains as before or grows more frequent and troublesome. If there was no expectoration at first or if it was scanty and mucous, it soon grows more abundant and may even become yellowish. At length, because of the obstinate character of the supposed bronchitis, or on account, perchance, of a streaking of the sputum with blood or, it may be, a slight but unmistakable hæmoptysis, a suspicion of the real nature of the difficulty is aroused, the expectoration is examined microscopically and bacilli are discovered. The lungs are now reëxamined and positive signs of pulmonary tuberculosis are detected.

In other instances the symptoms of bronchitis are associated with, or subordinate to, others of a general nature, loss of appetite, decline in strength and weight, an ashen-gray pallor that gives an appearance of anæmia, and the family doctor is consulted largely because of the increasing weakness. In these cases cough is rarely if ever absent but is a minor feature. On this account, mainly, the exact nature of the trouble is not suspected or, if surmised, is not definitely recognized. Tonics and a rest are ordered, and the individual is led to hope for a speedy return to health.

In the great majority of cases the signs of pulmonary involvement are indefinite, but now and then a case is seen in which the initial symptoms are trifling for a considerable time. Cough is present, to be sure, but expectoration remains scanty and yet, when the lungs are at length examined, the physician is surprised to find at one apex marked and even extensive dullness, distinct bronchial breathing and copious fine crackling râles. In such cases the constitutional symptoms are subordinate to the local signs, the reverse of what obtains in most instances.

Whatever be the precise mode of onset the disease is progressive. The temperature, which in the incipient stage was so slightly elevated as to have been thought normal, increases and the patient himself becomes conscious of the presence of fever, particularly in the late afternoon or early evening. Ranging in the beginning between 99.4° and 100.5° F. it now is found to fluctuate between 100.5° F. in the forenoon and 101.5° or 102° F. in the latter part of the day or after exertion. During the night sweating occurs, varying from slight moisture to profuse perspiration that wakes the patient from sleep or even necessitates a change of clothing.

Cough racks the sufferer, especially when his fever is at its height or it occurs at frequent intervals by day as well as by night. Expectoration is copious and either muco-purulent or entirely purulent, and microscopically it is seen to contain large numbers of bacilli mixed with pus-germs and occasionally elastic fibers or even fragments of pulmonary tissue. In this chronic form hæmoptysis may occur but is not apt to be present.

Dyspnœa is not noticed or is present only upon unusual physical exertion or after a fit of coughing. The pulse is accelerated, 100 to 120, small, weak and compressible. Emaciation becomes very apparent, anorexia is obstinate and may even amount to actual loathing of food, weakness is pronounced and nervous symptoms make their appearance.

The disease has now reached the stage of softening, is moderately advanced, and examination of the chest discloses well-marked dullness in one of the upper lobes with copious fine and coarse bubbling râles which obscure the breath-sounds. These signs are apt to be confined to one apex, reaching to the second or third rib in front and at the back to the lower angle of the scapula. Not rarely there are signs of incipient infiltration of the other apex.

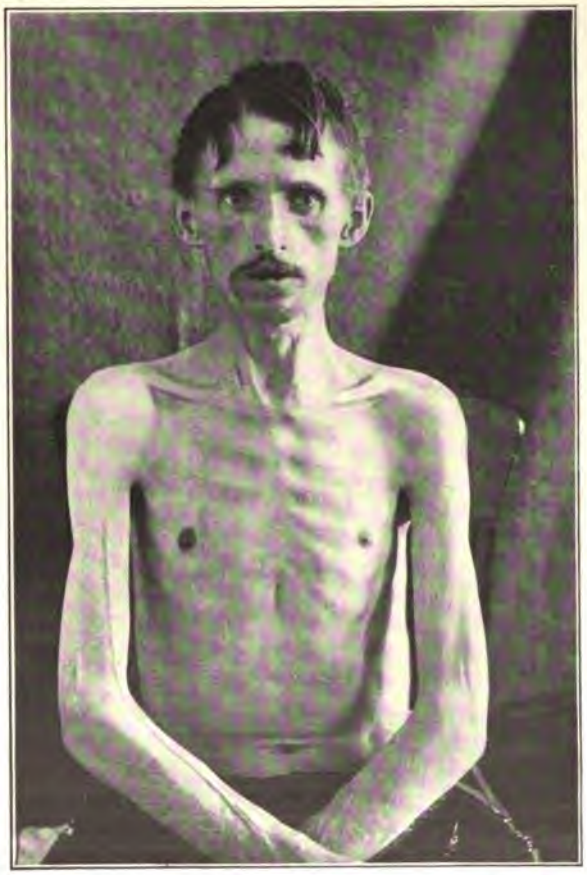


FIG. 67.—Characteristic habitus of advanced phthisis.

The chest is sunken, particularly on the side first affected, the shoulder muscles are wasted so that the shoulders droop forward causing the scapulæ to spread apart and producing the characteristic shape of the chest seen in Figs. 67, 68, 69. The sunken cheek with its spot of red in marked contrast to the general pallor, the bright eye, hollow chest and wasted musculature present the characteristic picture so well known and designated phthisical habitus.

Although in the course of the six or eight months that may have now elapsed the evidences of a wasting malady have become unmistakable even to a layman's eye, still the downward progress has not been steady. There have been periods of weeks when, owing to some new plan of treatment or a temporary change to the country, there have been encouraging signs of improvement. Appetite and digestion have improved somewhat, fever has decreased and strength has been gained.

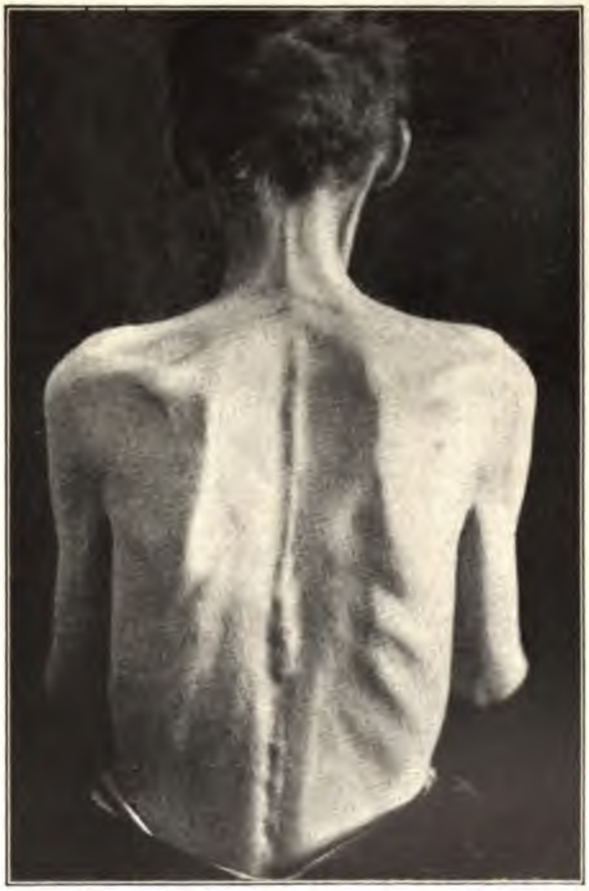


FIG. 68.—Posterior view of same chest as Fig. 67.

A faint color may have returned to the sunken cheeks which, filling out perceptibly, evince a gain in flesh. The cough and expectoration have not disappeared, however. After a time a fresh cold is thought to have been contracted and the cough increases in severity, or a sharp attack of diarrhœa pulls down the strength, or a sudden pain in the side with rise of temperature gives notice of a dry pleurisy. The gain, previously noted as a herald of returning health, speedily vanishes and is followed by a still greater loss in weight and strength.

If, up to this time, the patient may have still been able to drag himself out to business, he now finds his endurance totally inadequate and, although reluctant to give up, he is compelled to yield to the force of circumstances. he has lost the power of sustained application and makes only occasional attempts to remain at his post. In most cases, with the oncoming of the stage of excavation the decline in physical powers has become so great that a state of pronounced invalidism has been reached. The patient remains in the house altogether or drags himself out only on warm sunshiny days, coughing and short of breath with the effort.

Examination of the lungs now shows that both have become affected or that the one primarily involved is more or less dull throughout. In the upper lobe are signs of cavity more or less distinct, while below are tubular breathing and numerous fine and coarser clicks, especially after cough. The opposite apex is in a state of softening, the heart is rapid and its sounds are feeble or accompanied by an anæmic systolic murmur.

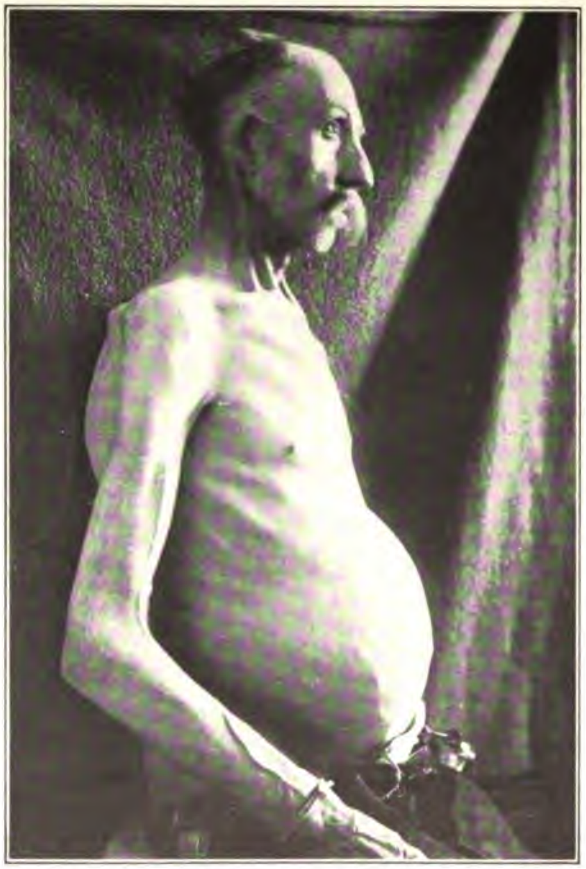


FIG. 69.—Pulmonary tuberculosis and tuberculous peritonitis.

The cough and expectoration have changed somewhat in character. In the early morning and occasionally through the day the cough is somewhat paroxysmal and brings up with greater or less difficulty purulent nummular sputa. At other times the cough is frequent, short, and unproductive, indicating an annoying sense of irritation. The voice is now likely to have grown husky and feeble, in some cases distinctly hollow or in others hoarse or even whispering from complicating involvement of the larynx. The bowels are persistently loose, or constipation alternates with periods of diarrhœa. The fever has become distinctly intermittent, in the morning the temperature being 97° F. and in the afternoon 102.5° or even 104° F. Its rise is preceded by a chill or chilly sensations, and its decline is succeeded by a marked, often drenching sweat.

Wasted to a mere skeleton, too feeble to sit up, shivering with cold at the slightest draught of air, able to take but scanty amounts of food and often racked by cough, the consumptive remains in bed or, possessed by the idea that his shortness of breath and palpitation are due to weakness of the heart, he still drags himself out of bed and downstairs each day. Some of these patients even persist in a daily walk when the weather is fine, although extreme weakness and shortness of breath compel frequent rest.

Notwithstanding the obvious progressive decline the patient still hopes, nay believes, he is going to get well, and may even plan new undertakings, journeys or other equally impossible proceedings. In only too many cases the consumptive who is plainly not far from his end becomes possessed with the desire to seek another climate in which he may recover from his "bronchial difficulty," or, as is too often the case, he is sent away on the advice of physicians or friends. The journey is made with great fatigue and difficulty to Phœnix or elsewhere; for a brief time the warm sunshiny weather and the stimulus of new hope keep him out of doors and a flickering rally is made. The improvement is shortlived, however, and a few weeks, or at most two or three months, after his change of climate the poor deluded and misguided mortal returns home in his coffin.

The foregoing picture has many variations in outline and coloring. In one case the onset is so insidious that no definite time of its beginning can be given. Almost without being conscious of his decline the man finds himself coughing and expectorating. Having lost a brother with consumption and now recognizing his symptoms as similar, he consults a physician and is scarcely surprised to learn that his left lung is diseased in its upper third. He arranges his business so as to spend the forthcoming Winter in the South, and in the Spring he returns much stronger and stouter, but still coughing and expectorating. The gain is held until Fall and hence he decides to stay at home this second Winter. Soon after the holidays, however, he finds he has taken cold and is not so well. Consequently he goes to California until May, and is again gratified to find the change has done him good.

This time he finds his weight is a little lighter and his strength is not quite so great. Examination shows considerable extension of the original disease, but dullness and bronchial breathing supersede râles. There is moderate fever, perspirations are not severe, appetite is fair and, aside from considerable morning cough and expectoration, the symptoms are not very troublesome. The heat of Summer is felt to be more depressing than usual and in dusty weather the cough is more frequent. On the whole, however, this second Summer passes and the condition does not seem to have grown materially worse.

In one of his business trips to a neighboring city a physician is consulted who has a local reputation for successful treatment of lung diseases. A new tonic is prescribed or the malt extract, cod-liver oil and hypophosphites are continued and in addition the importance of a change to a suitable climate is emphasized. The advice is heeded or not according to the condition in which the patient finds himself when cold weather sets in and as business makes it convenient or otherwise. Should he not go away, and should the Winter happen to be mild, he gets along fairly well. In the worst weather he stays in the house and, on the whole, he passes the cold months with, as he thinks, only a little change for the worse.

In this manner five or even eight years may elapse and still the invalid is able to go to his office on most days. He looks thinner, paler and more feeble, while his cough is more frequent and his expectoration is thick, yellow or greenish-yellow. At length, just as he is about to leave the home climate in December he is seized with a severe cold on his chest, pneumonia sets in and he is carried off in a few days.

In other instances, after the pulmonary disease has progressed for a year or eighteen months a sudden aggravation of all the symptoms occurs. In particular the fever is increased, the cough is frequent and severe, and it is found on examination that an acute miliary tuberculosis has supervened, or that the previously intact lung is now the seat of an acute diffuse tuberculous pneumonia. The patient fails rapidly and dies in four to six weeks.

In another class of cases the history is something as follows: A man of about forty, whose brother was living in Colorado on account of his lungs, was seized one morning on walking to the cars with a sudden hæmoptysis in which he lost about two ounces of blood. He sought his family physician and by her was referred to a specialist. The history was as given above and in addition the man admitted that for a long time he had had some cough with a little morning expectoration. There was very slight rise of body-temperature and the pulse was not especially rapid nor of low tension. On examination of the chest the physician was surprised to discover dullness at the right apex to the third rib in front and mid scapula behind. Below the middle of the clavicle was a small cavity and over this area some fine bubbling râles were heard.

The man was ordered home and to bed without delay. To make a long story short, repeated profuse hæmorrhages occurred in the course of the next two weeks which greatly reduced the strength. Fever became continuous and râles were audible at the base of the right lung in front, with dullness and feeble breath-sounds in the back. At length there came a sudden profuse flow of blood from the mouth and the patient died in a few minutes from suffocation.

In 1889 a healthy looking girl of nineteen was seen on account of epileptic fits. Her brother was under treatment at the time for advanced consumption of which he died a few months later. Not long thereafter the sister was lost sight of. In the early Summer of 1903 she was again seen and gave the following history: She was married and had one child of some five years. She was still a sufferer from epilepsy which she was able to hold in check by the continuous use of bromides. She had been having trouble with her lungs for several years and the Winter of 1902 and 1903 had been in Phœnix. There she had greatly improved, but on returning home had been advised by Dr. Craig to place herself under my care.

The left lung was extensively diseased, being dull practically throughout and showing signs of a large vomica. Fine bubbling and subcrepitant râles were everywhere audible except over the location of the cavity where amphoric breathing and cavernous voice were present. Her temperature was normal, but her pulse was rapid and weak. Cough was not very frequent, expectoration was thick and purulent. Her appetite was only moderate, but emaciation was not marked. Her spirits were depressed.

She was seen at regular intervals and she was encouraged to take as much outdoor air and nourishing food as possible. Ichthyol was given to control the expectoration. By Fall her condition had still further improved and the râles had disappeared, except in immediate proximity to the vomica. On two or three occasions she had shown a slight transient rise of temperature. In the Fall of 1903 she was sent to Asheville in care of Dr. C. L. Minor and there she remained until June, 1904, improvement being gratifying until shortly before her return home.

When she reached here her condition was not good; the temperature ranged from 100° to 101.6° F., P.M., her weakness was extreme, perspiration was annoying and cough was troublesome. The left lung showed the old signs, râles being numerous, and in addition there were signs of early involvement of the right upper lobe.

She was kept at rest in bed on a porch and as good care was given as her rather limited circumstances permitted, with very little medicine save what was necessary for relief of symptoms. Improvement slowly followed, but she remained weak and discouraged. She had lost hope. In October, 1904, she was again urged to go to Asheville, but objected on the score of her extreme sensitiveness to cold weather. Instead, she desired to go to El Paso, where she could enter a sanitarium kept by a Catholic Sisterhood. She has not been heard from since her departure.

In her case the process was chronic and characterized by loss of strength and mental depression rather than by marked wasting, septic temperature or harassing cough. Appetite was poor and sputa were purulent, but toxæmia was not a marked feature.

However varied the clinical picture in detail, in leading features it is ever the same: insidiousness of onset, slow destruction of pulmonary tissue, gradual, progressive wasting and decline of strength, fever, cough, expectoration. Periods of improvement alternate with periods of relapse, but gradually and surely the signs of disease grow more apparent. Months drag on into years before the emaciated frame finally surrenders its hold on life, or the slowly downward course becomes suddenly hastened by some intercurrent catastrophe as fatal hæmoptysis, pneumothorax, acute general miliary tuberculosis or acute miliary tuberculosis limited to the lungs.

In my service at Cook County Hospital at the present writing is a male patient with right-side hydropneumothorax who states he has had cough for two years. In the right upper lobe are dullness, bronchial breathing and a few indistinct râles, while at the base is drumlike tympany surmounting an area of flatness. This line of absolute dullness shifts with change of position, and over this area can be obtained the succussion splash and the coin sound. The man was, apparently, not especially ill until the sudden occurrence of dyspnœa announced the development of the pneumothorax. He now maintains the left lateral decubitus, has continuous fever, profuse perspiration and extreme prostration.

A female patient was recently admitted to Ward 6, Cook County Hospital, on account of cough, pronounced dyspnœa and high fever. No very clear history was obtainable from the patient who merely stated that she had gone to work in a laundry as usual about a week before. There she had been taken with a chill and become so ill she had to return home.

The physical signs were cyanosis, rapid, labored respirations of about 60 to the minute, very frequent feeble pulse and throughout the entire chest a multitude of fine and coarse bubbling râles which entirely obscured the breath-sounds, while only at the right base behind could dullness be detected. In general the percussion-note was somewhat tympanitic.

The diagnosis hung between acute pulmonary tuberculosis and a pneumococcus infection with widespread bronchitis and areas of lobular pneumonia. I favored the latter because of the history of its sudden onset a week earlier. It was ascertained after the patient's death, however, that she had been in poor health and had had a cough for two years.

Death occurred a few hours following my single examination and at the autopsy the case was found to be one of tuberculosis. In both apices were small old vomicæ, while the lungs were completely studded with minute grayish nodules and areas of broncho-pneumonia that, in the right lower lobe, were so closely set as to give the appearance of a diffuse infiltration. The tubes were filled with purulent secretions.

January 11th, 1905, a young married woman was admitted to the same hospital, in the seventh month of pregnancy. She had cough and fever, and examination of the lungs revealed dullness, bronchial breathing and fine crackling râles in the right upper lobe. The balance of the lungs, the heart and all other viscera were negative. From the history and physical signs the case was put down as one of chronic pulmonary tuberculosis with pregnancy, and microscopic examination of the sputa later verified the diagnosis.

January 17th the woman aborted, a dead fœtus being expelled. The placenta appeared to be intact. Soon thereafter the patient's condition grew worse, temperature became high and irregular, and respirations were greatly accelerated, out of all proportion to the pulmonary findings which appeared essentially as on admittance. The case was now regarded as one of acute miliary tuberculosis of the lungs. I did not see the patient but am told the heart was not again carefully examined. Death occurred three weeks after her admission to the ward.

The autopsy disclosed a most interesting and unexpected combination of conditions. The right upper lobe was the seat of an old cavity with diffuse tuberculous broncho-pneumonia in a state of softening, while the remainder of that lung and the whole of the other were thickly studded with miliary nodules of recent formation. Similar gray masses were also scattered throughout liver, spleen, kidneys and peritoneum while the small intestines contained tuberculous ulcerations.

Surprise lay, however, in the discovery of an added puerperal infection. In the cavity of the uterus was a small fragment of broken-down placenta while the tubes contained pus which could be squeezed out and which explained a few drops of pus found in the pelvic peritoneum. The aortic an mitral valves were the seat of recent soft thrombi from which emboli had been carried into the spleen and the liver, so that in these organs there were septic infarcts alongside of miliary tubercles.

Had death been postponed it is probable that the endocarditis would have been detected *intra vitam*, but as it was, the intensification of symptoms was attributed to the miliary tuberculosis, diagnosis of which had rested mainly on the dyspnœa together with the recognition of an old process in the right upper lobe. Whereas the miliary nodules so thickly disseminated in both lungs had contributed to the rapid respiration-rate, it is likely that the endocarditis also was a factor in its production. The complication disregarded, the case illustrates how suddenly and radically the course of a chronic phthisis may be altered.

Although cases of pulmonary tuberculosis may resemble one another in the essential phenomena of cough, expectoration, fever and emaciation, still, in details, no two are alike. Some which begin slowly and insidiously may be suddenly terminated in the manner just described, while others may now and then be seen which, commencing acutely with rapid and even extensive destruction of lung-tissue, yet seem to rally after a portion of the lung has been cast off, and thereafter pass into a slowly progressing condition and manifest the characters of ordinary chronic ulcerative phthisis. They may drag on for months or years with signs of cavity, more or less profuse purulent expectoration, a low grade of fever with periods of encouraging improvement alternating with times of discouragement.

Occasionally a case is seen in which, after months of failing health characterized by growing weakness rather than by pronounced cough and fever, a sudden change for the worse is experienced. The evening temperature is found to be 102.5° F. and the patient is compelled to take to bed. Signs of localized pneumonia appear at one apex, cough is harassing and after a few days there is a sudden expectoration of a large amount of yellowish sputum containing bacilli and pulmonary tissue. A pulmonary abscess has formed and been evacuated leaving signs of cavity behind.

The fever abates *pari passu* with amelioration of cough, appetite returns, strength and weight increase, and in the course of a few weeks more the invalid is sent to a more favorable climate. Here the gain is still more rapid and after the lapse of six to nine months he thinks himself well enough to return to his home. Thereafter one of two things occurs: either he rallies sufficiently to get an arrest of his disease, or, after a period of improvement in which he regards his enemy as practically defeated, he is surprised by a fresh attack on the part of his ever-lurking foe and henceforth wages a steadily losing fight to the end.

Still another tuberculous subject has a cough which he does not regard as serious. He expectorates but looks upon it as bronchitic, loses somewhat in weight and strength but still keeps at his daily work. At length, without warning, he is seized with a sudden sharp pain in his side and, not able to breathe or cough, seeks his bed or admittance to some hospital. Examination now discloses signs of pneumothorax to which fluid is soon added. The patient lies on the affected side in bed, with high irregular fever, sweats profusely, breathes and speaks with difficulty, coughs and expectorates with still more difficulty, fails steadily, is the object of much interest on account of his clinically instructive signs and at length dies, hardly aware of the fact that he had for many months been the victim of a relentless insidious disease. Such, in brief, was the history of a negro assigned to my service at Cook County Hospital in the Winter of 1905.

Still another phase of this terrible disease was shown by the case of a brilliant young physician some ten years back. While serving his interneship he was attacked with pleurisy on the left side with effusion. Its ætiology seemed amply attested by the presence of tuberculous glands on the corresponding side and he was advised to seek change of climate. He left directly for the Southwest and spent the next two years in various places, but not under the most favorable conditions, as he persisted in attempting to practice whenever and wherever he found opportunity.

For the first months his health improved in general although the local disease made progress. I saw him and examined him at Las Vegas, N. M., in December, 1896. He was in fair flesh and displayed but slight cough, but his expectoration, what there was, was purulent and he had a moderate afternoon temperature. The chest examination showed extensive disease of the right lung, dullness, bronchial breathing, a few râles and signs of small cavity in the upper lobe, while in the middle lobe was a secondary broncho-pneumonia that was leading to destruction of that portion of the lung also. The left lung was not so much nor so seriously affected, to judge from physical signs, although it was the one primarily involved.

The next year was dragged out in very much the condition described, excepting that he suffered from an obstinate diarrhœa, probably tuberculous, and he slowly became incapacitated for work. At length he brought up in Oracle, Ariz., where he was too feeble to take much exercise. By this time, I understand, his left lung had become extensively invaded by the ulcerative process and finally death resulted from a profuse pulmonary hæmorrhage.

It is apparent that the course and characters of the pulmonary process are determined by the predominant characters or tendencies to softening or arrest displayed by the tuberculous infiltration in any given case. For this very reason it is difficult always to classify cases with respect either to their chronicity or the precise morbid anatomical characters within the lungs.

I am speaking now of chronic pulmonary tuberculosis and yet, such are the differences in duration that, excepting in well-marked instances either of protracted course or of acute progress, it is not always easy to classify cases as chronic or acute. In a similar manner it generally seems to me folly or an attempt at over-refinement to say whether a case is fibroid, caseous or fibro-caseous, as is done by Fowler, e. g.

Occasionally a case is encountered which displays such pronounced tendency to caseation and formation of cavity that it may be spoken of as caseous. The process manifests no tendency to repair, but the broncho-pneumoni foci break down rapidly and form cavities. Others again show signs of ir duration with slow destruction, but no retraction to speak of and vomi appear gradually and enlarge slowly. These may be said to be fibro-caseo since they stand in an intermediate position between the former and those that are distinctly fibroid.

In this last the tendency is to slow hardening and retraction of the lung with few signs of softening. One is unable to say definitely whether the vague signs of cavity point to true vomica or to bronchiectasis. The local signs of softening are not marked and the constitutional symptoms are not those of well-marked toxæmia. Consequently it may be possible in exceptional instances to put these patients into a class by themselves.

As a general proposition, however, it is sufficient and wiser merely to content oneself with the attempt to recognize cases of pulmonary tuberculosis as such. The clinical pictures may vary in detail but in main features they are ever the same. They may vary in beginning, present variations in severity and duration, terminate in different manners, but they are all pulmonary tuberculosis with more or less mixed infection and may be dealt with as such.

Modes of Onset.—These are numerous and widely different. (1) The most usual manner of beginning is as a *bronchitis*. This may develop slowly and so insidiously that the individual cannot state the date of its commencement, but in most instances it is supposed to begin as a cold. The patient fancies he has been exposed and says his cough began directly thereafter. It is generally possible, however, in such a case, to elicit a history of slight cough for a few weeks previously, so that it is clear that the fancied cold was in reality a sudden exacerbation of a preëxisting bronchitis.

(2) The individual begins to notice a slow but sure *decline in strength*, and either has no cough or this is an unheeded dry hack. His weight also falls off, but so gradually as not at first to attract attention. He feels tired and listless, "run down" as he says. In a female teacher of twenty-five seen recently the loss of strength began in August, but it was not until the next December, after having resumed her school duties for nearly three months, that she was finally forced to give up teaching. Then it was noticed that she had a cough and slight rise of temperature.

Examination of lungs was made and bronchitis was diagnosed. This did not yield to remedies and on her doctor's having the scanty mucous sputum examined microscopically bacilli were found. A week prior to my seeing her she had a slight hæmoptysis. Although she stated she had gained in weight it was possible to discover signs of early involvement of the left upper lobe; dullness in the left supraclavicular and suprascapular regions, impairment below the inner end of the clavicle, a circumscribed small patch of more apparent dullness in the second interspace below the outer third of the clavicle, where, according to Fowler, an area of secondary involvement may occasionally be detected. Over all these areas the breath-sounds were feebly bronchial, but there were no râles.

(3) In some cases the disease is masked by *dyspeptic symptoms*: anorexia, that may amount to actual repugnance for food, distress after eating and sometimes vomiting. These cases may closely resemble gastric ulcer and are very apt to be treated for weeks or even months as cases of nervous dyspepsia if not ulcer. I recall a young woman whom I treated many years back in

whom the lavage brought up large amounts of mucus with relief, but who, nevertheless, was not able to eat, and who subsequently showed plain signs of pulmonary tuberculosis.

(4) *Hæmoptysis* is in some instances the earliest symptom calling attention to the lungs. This was so in a young man, a bookkeeper, who, when I saw him shortly thereafter, showed slight dullness, bronchial breathing and indefinite râles at the right apex. He was sent to New Mexico, improved immensely, returned to his desk, and in time displayed marked aggravation of his general and local symptoms which necessitated his going away for a second time. His subsequent history is not known.

(5) In some cases the onset is first declared by a *pleurisy*. This may be dry and without definite signs of disease of the corresponding apex, but I have known it to be with effusion. A case in point was that of a young man brought to me by Dr. Parsons, of Ravenswood, Chicago, because of pain in the right side, shortness of breath, pronounced dullness at the right base and fever of 101° F. The signs of effusion were present, dullness from level of third rib downward, feeble bronchial breath-sounds, diminished vocal fremitus and slight displacement of the apex-beat to the left.

Twenty ounces of clear serum were drawn off without making any difference in the physical signs, and fever persisted. Cough was slight without sputum, and after a few days the patient declared himself able and anxious to get up. As things did not improve aspiration was again made with the same result, 20 ounces of clear serum and no more. A year subsequently his left apex was found to show signs of incipient disease and a year later he died in Asheville, N. C., as I was informed.

The pleuritic friction may be discovered at an apex or at the base. The former class of cases is likely to arouse suspicion of the real nature of the process in the mind of the practitioner, but when the pleurisy is at the base it is not always regarded as tuberculous. Nevertheless in nearly all instances of pleuritis with effusion that begin insidiously the process is of this nature. The involvement of the pleura may be declared by acute symptoms. It was the fact that the pleurisy may precede the onset of pulmonary tuberculosis by years which led Leming and others to maintain the view that the subsequent lung-involvement was the result of mechanical interference by adhesions with proper expansion of the lung.

(6) In some not very frequent cases the disease commences as a *laryngitis*. The voice becomes husky and cough is frequent and intractable. The larynx generally shows infiltration in the interarytenoid space or of a cord and examination of the lungs may disclose dullness and modified breath-sounds at one apex. These cases are most important and should be diagnosed early.

(7) Osler calls attention to the fact that the pulmonary disease is son times preceded for years by *cervical adenitis* and I have recently seen su a case. A woman of forty-nine was examined by me in Muskatine, Iowa, account of cough and expectoration suggesting bronchitis or incipient phth On the right side of the neck was a large mass of lymph glands, evid tuberculous, which had existed for six years. The corresponding apex be

was impaired, but in the left apex were unmistakable though slight signs of early mischief, dullness, feeble bronchial breathing and crumpling. Her temperature was 100.4° F., but notwithstanding the existence of cough for a year she had not lost much in weight.

(8) Osler directs attention also to a class of cases which he calls latent. They are seen in laboring men chiefly and one is often surprised by the discovery of local signs out of all proportion to the degree of constitutional disturbance. There may even be a small cavity. I do not now recall such an instance.

(9) In examining cases of chronic pulmonary tuberculosis one not infrequently obtains the history of the illness as having followed upon typhoid fever. Such a case was seen very recently, but as a Widal test was not made at that time there seemed to me a reasonable doubt as to the exact nature of the fever. I certainly have seen cases in which I was sure that the supposed typhoid was in reality a manifestation of the tuberculous process. We all know how readily acute general miliary tuberculosis may be mistaken for enteric fever, and I see no reason why a pulmonary tuberculosis, beginning with unusually pronounced reaction but afterwards running into a chronic course, may not likewise lead to erroneous diagnosis.

Analysis of Symptoms. *Temperature.*—An increase of the body-temperature is probably one of the earliest indications of incipient pulmonary tuberculosis and hence, as emphasized by all writers, careful record of the temperature should be kept in every suspicious case. As long as the process is unmixed, i. e., purely tuberculous, the body-heat is raised from one to two degrees, ranging from 99.5° to 100.5° F. The patient may be wholly unconscious of his fever or, on close questioning, may admit that toward evening he is aware of a sense of increased warmth, shown by dryness and heat of the palms or by a feeling of thirst. As stated by Fraenkel the evening rise of temperature may come on as a sudden flushing of the face or of the whole body.

In these cases I have often noted that the coldness of the hands is in marked contrast to the heat of the skin underneath the clothing or of the chest when bared for examination. It is also worthy of note that the temperature is higher after than before the examination of the lungs. This is in accord with the well-known observation that the temperature of tuberculous individuals is easily elevated by a degree of emotional excitement or physical effort that would not so affect a healthy person (Pensoldt). Not infrequently the patient complains of annoying, even profuse, perspiration at night.

The maximum increase is not always in the evening, although such is common, but may take place at noon or soon thereafter, and hence the thermometer should be used at regular intervals during the day in all doubtful cases. It is my custom to have the temperature recorded at nine, twelve, three, six and nine o'clock. If, instead, it is taken only twice daily, morning and evening, the increase may be readily overlooked. Fraenkel states a fact which is important but was, I must frankly admit, not previously known to me, namely, the monthly temperature-curve of tuberculous women is apt to reach its highest point at the time of the menses and may be seen to rise gradually before and decline gradually after the catamenia.

As remarked, the fever of the incipient stage is so slight as to be easily overlooked by practitioner as well as patient, but there are cases in which it presents a distinctly remittent character. When, in addition, there are chills or chilly sensations and perspirations the case may be mistaken for one of malaria. It is this fact that led Osler to mention one mode of onset as being with symptoms highly suggestive of malaria. In such instances a false diagnosis may be guarded against by a blood examination for the detection of the plasmodia.

The cause of this low grade of fever is probably to be found, as suggested by Cornet, in the absorption of toxins generated by the bacilli. Before Koch's discovery it was considered a symptom of inflammatory action, and Fraenkel expresses the opinion that the eruption of tubercles in the lungs may be attended by febrile reaction just as much as in the case of tuberculous inflammation of the pleura or other serous membranes.

Whatever be the explanation of the phenomenon, it is established that so long as the process is purely tuberculous the fever is slight in chronic cases. Indeed there are instances in which the temperature remains practically normal and hence some physicians go so far as to declare that fever is not a manifestation of tuberculosis *per se* but of an added infection, i. e., a mixed infection. As we shall see, this is probably correct in the stage of softening, but in the incipient or unmixed stage, the elevation of temperature is attributable in some manner to the action of the bacilli. In support of this proposition may be cited the observations of Schabad who has shown that tuberculosis without the admixture of any other germs is capable of producing high temperatures, such as are generally attributed to streptococci.

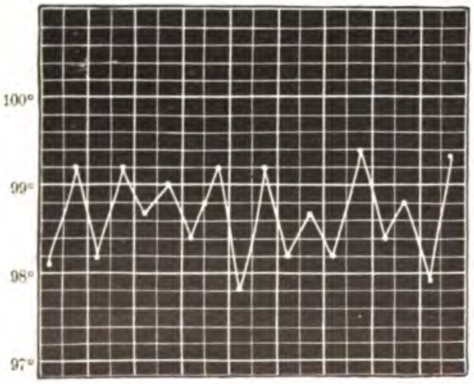


FIG. 70.—Chart of temperature characteristic of early pulmonary tuberculosis (Struempell).

In the stage of softening, that is, with the advent of muco-purulent or purulent sputa and the detection of larger moist râles in the lungs, the body-temperature assumes a higher general average and a somewhat different character. It is now a remittent or intermittent fever whose minima fall in the fore part and the maxima in the after part of the day as before. The range varies in different cases; in the remittent for the fever may be said to remit 100.2° to 100.5° F., while its ma ma may reach 102° or 102.5° F. the afternoon or evening. During the forenoon the hands are cool and patient may not be conscious of having fever, but along about midda experiences slight chilliness or even an outspoken chill and soon there becomes aware of considerable elevation of temperature.

There is much diversity, however, according to my observation, in the subjective sense or consciousness of fever-heat on the part of these tuberculous individuals. Some will tell you that they have fever and will show it by increased excitability and perhaps slight trembling, but, as a rule, I find that the sense of augmented body-heat is subordinate to other more annoying symptoms as cough, weakness, etc. The effect of physical exertion on the temperature of the body is also noticeable, inasmuch as the fever is maintained at a higher general average by the patient's keeping up and around and the period of remission may be curtailed by the physical effort of a trip into the city or to a physician's office. This fact has its bearing, of course, upon treatment, as will be shown in the proper place. The temperature at this time is shown by the accompanying chart (see Fig. 70).

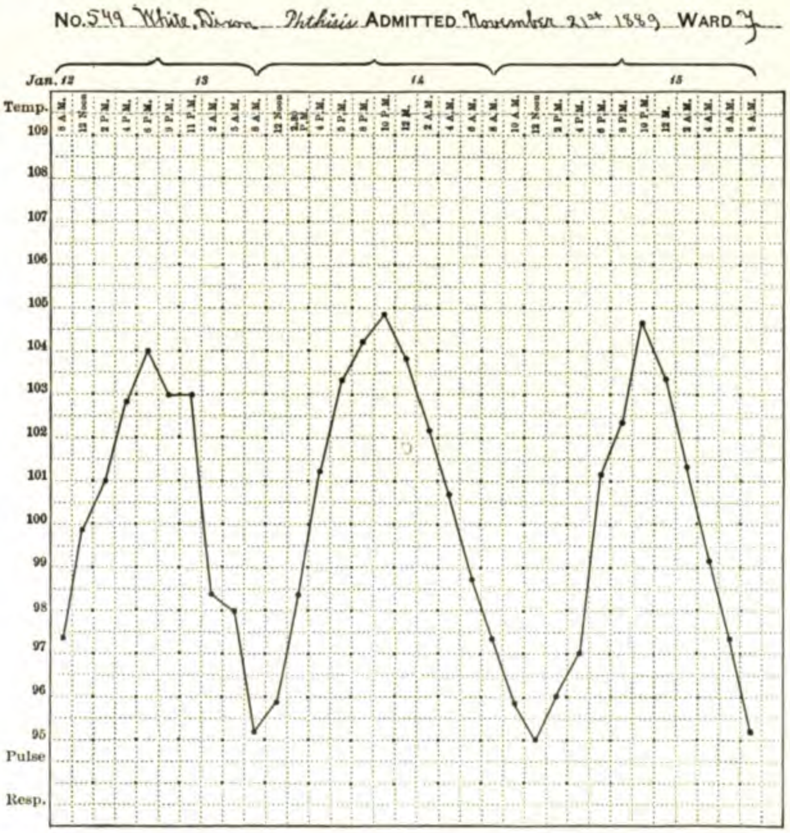


FIG. 71.—Chart of temperature characteristic of stage of excavation (Osler).

As softening progresses and especially as excavation grows apparent the extremes in the temperature become more marked and what was before a remittent is now an intermittent type of fever, the so-called *hectic fever.* The thermometer registers normal or subnormal in the morning, and in the afternoon or evening 102°, 103° or even 104° F., according to the intensity of the toxæmia. The rise and fall of the temperature may be regular or the pyrexia may display an irregularly intermittent type, as seen in the annexed chart (Fig. 71).

This hectic fever is generally believed due to a mixed infection, commonly with the streptococcus pyogenes, but concerning its exact causation there is still some divergence of opinion. Schabad has reported a case in which the temperature was distinctly hectic, and yet careful bacteriological investigations, both before and after death, demonstrated conclusively that the only germs present were tubercle bacilli. Nevertheless this was but a single instance and does not disprove the generally accepted fact that in advanced consumption other bacteria than the bacilli are present both in the sputa and in the caseous foci.

In a few instances there may be a veritable bacteræmia as proven by blood cultures, but in the main the hectic temperature is thought due to absorption of toxins generated by the streptococci in the lungs. For this reason Cornet speaks of it as the *fever of absorption.* It is a remarkable fact, however, that even when the expectoration is purulent and cavities exist there is not always pyrexia. Indeed, cases of advanced phthisis are often seen which display no rise of temperature for weeks and even months. I have seen numerous such instances at the Cook County Consumptive Hospital. The patients are generally much reduced in weight and strength and display signs of consolidation with excavation but not progressive softening. They are apt to expectorate large amounts of purulent sputum. It may be that the purulent material comes from the cavity and hence the absence of hectic phenomena. To this point we shall return anon.

Spengler distinguishes two types of mixed infection, namely, active and passive. In the active the lungs are invaded by both tubercle bacilli and streptococci, often in enormous numbers, while the sputum may contain no other than the last-mentioned organisms. The patients display chills, sweating, anorexia emaciation and other phenomena of pus infection. In the passive form hectic fever is wanting and the sputa may contain no streptococci or these only in small numbers.

Petruschky explains the presence or absence of streptococci in the sputa and the corresponding decrease or increase of the fever on the ground that the discharge of pus is favored or hindered. As long as the contents of a vomica are freely evacuated there is little or no absorption, but when the communicating bronchus is blocked the purulent material is retained, as shown by disappearance of streptococci from the expectoration. Consequently a type of fever results which is characterized by wide differences in its extremes and which the Koch school has termed the Streptococcus curve (Fraenkel). That such a view does not suffice for the explanation of all the observed facts connected with the phenomena of hectic fever seems proven by the case of Schabad's, previously cited, and by the investigations Schroeder and Mennes.

These investigators came to the conclusion that the streptococcus obtain from phthisical sputa is far less virulent than is the same organism deriv from erysipelas or septic processes. Intravenous injections of the filtrate of liquid cultures, and even of the cultures themselves, produced in animals no other results than slight febrile reaction. Accordingly Schroeder and Mennes came to the conclusion that no direct or indirect connection could be shown between the hectic fever of chronic tuberculosis and the streptococci or their toxins.

The question cannot be regarded as settled, however, by the experiments of the above-mentioned investigators. It is not a fortuitous circumstance that pyogenic bacteria appear in the sputa at or about the time when the temperature ceases to be a mild remittent or low continuous one and assumes a more or less pronounced hectic type, while at the same time signs of breaking down of the lung appear. Indeed, Prudden's experiments seemed to demonstrate an intimate causal connection between the introduction of streptococci into the lungs of tubercularized animals and softening. It is, moreover, a common clinical observation that the more active the breaking down of pulmonary tissue, the more intense are the fever, chills, perspirations and emaciation, so that one cannot escape the impression that such phenomena are dependent upon the mixed infection and not upon the tuberculosis *per se.*

This conclusion appears to be strengthened, moreover, by Kerschensteiner's elaborate bacteriological studies. He investigated various forms of pulmonary and bronchial disease, nontuberculous as well as tuberculous, that were attended by formation of pus, and found in all alike several varieties of pyogenic organisms, but chiefly streptococci, thus corroborating the work done by Schabad and others. In the light of such facts the conclusion is warrantable, I think, that when fever is wanting in chronic ulcerative phthisis the bacteria have lost much of their virulence or the absorption of their toxins is prevented in some such manner as suggested by Petruschky.

Whatever be the precise explanation it may be stated, in passing, that from a prognostic standpoint the absence of fever is favorable, and yet, without febrile temperature, it is possible for the pulmonary disease to make progress. It is this fact that has led some authors to speak of a toxæmia in cases of consumption without pyrexia. The toxæmia in such cases is shown by increasing weakness, emaciation, perspirations, anorexia, etc. In such instances the morning temperature is apt to be persistently subnormal while that of the afternoon is normal or subfebrile. Consequently, in estimating the value and significance of fever-free periods in these chronic types of pulmonary tuberculosis one must take into consideration all the attendant phenomena.

Emaciation.—One of the very earliest symptoms of this disease is progressive loss of weight. It is not present to the same degree in all cases, yet is never wholly wanting. It is so early a feature that in some incipient cases it is the first to attract attention, and the loss of flesh may even proceed with such rapidity as to be in striking contrast to the small amount of lung-involvement. Other individuals with pronounced signs of early infiltration of an apex may yet display but slight or moderate emaciation.

Such differences render it plausible, I think, that several factors are responsible for this early loss of weight. Of these the most important, probably, as stated by Cornet, is a toxæmia resulting from the action of the bacilli within the lungs. This view is strengthened by the observation that many of the victims of incipient pulmonary tuberculosis lose weight out of all proportion to the degree of the fever, and in spite of their retaining fair appetites. Some persons, moreover, begin to emaciate before they manifest other noticeable symptoms, while indeed they are still in what may be termed the latent stage. Strictly speaking their disease is not latent if it have already caused any symptom whatever, but the term is yet a convenient one if we wish to distinguish this very early period from that in which more decided symptoms begin to annoy the patient.

In other cases the emaciation seems to depend, in a measure at least, upon the persistent though slight elevation of temperature, and in others again upon the obstinate anorexia and want of assimilation. In still other individuals there is such a degree of anæmia that this probably contributes to the loss of weight. On the other hand, it may also be asserted that these contributing factors are themselves but manifestations of the tuberculous infection, and hence it is a begging of the question to attribute the emaciation of the incipient stage to anything else than a toxæmia.

Such is not wholly correct, however, since it is a matter of everyday observation that with the removal of all contributing factors a gain in weight generally takes place. On the other hand, in the advanced stage, when hectic fever sets in, emaciation proceeds with increased rapidity; so that at this time we are forced to recognize fever and lack of sufficient nutrition as powerful elements in the production of this striking phenomenon.

The emaciation affects not only the subcutaneous fat but involves also the musculature, and this latter fact probably accounts in part for the extreme debility seen in advanced cases. Toward the end of the disease, provided death does not result from hæmorrhage or some other intercurrent complication, the wasting becomes so marked that the consumptive looks like a mere skeleton, a shadow of his former self. Indeed, it is this striking tendency to emaciation that gives to pulmonary tuberculosis the popular name of consumption.

Increasing loss of weight is generally thought to denote progress of the pulmonary disease and gain in flesh a tendency to arrest of the local process. This latter is not altogether true, however, for cases are now and then encountered in which the patients will show some increase of weight while the pulmonary signs do not show appreciable change for the better. Such was true in a young Bohemian with extensive involvement of the left lung whom I had under observation in the Summer of 1904. Flesh and strength were gained, cough and expectoration were somewhat lessened, but the dullness and copious râles did not become less marked.

Increase in weight, or at least an arrest of the wasting, may sometimes be observed under a system of forced feeding even when the temperature still shows slight febrile elevation, but, as a rule, marked gain in weight does not take place until the individual is permanently free from pyrexia. Yet, on the whole, progressive emaciation is to be regarded as an unfavorable and the taking on of flesh as a favorable feature of the case.

Respiratory System.—Dyspnœa cannot be regarded as a legitimate symptom of chronic pulmonary tuberculosis apart from some complication, as emphysema, pneumothorax or pleuritic effusion, that interferes with hæmatosis or occasions pronounced cardiac inadequacy. Indeed, I have seen persons with such extensive destruction of their lungs that they might literally be said to have only half a lung left and yet they did not manifest or complain of shortness of breath. Such cannot be said to be the case in acute miliary tuberculosis of the lungs, for in such a case there is often a marked objective dyspnœa, shown by rapidity of breathing.

One sometimes observes consumptives who, in spite of extensive destruction of lung-tissue and consequent reduction of respiratory capacity, display far less frequency and difficulty of breathing than would be naturally expected. They may exhibit objective increase of respiratory rate and yet make no complaint of subjective dyspnœa. I have been accustomed to attribute this absence of air-hunger to a decreased production of carbonic acid, but in the light of M. Albert Robin's investigations such an explanation is inadmissible. His observations and conclusions are as follows:

By an ingenious and simple arrangement of rubber bags and tubes the expired air is collected and is then subjected to careful chemical analysis. From a long series of trials under carefully controlled and uniform conditions M. Robin arrived at the following facts: The production of CO_2 by tuberculous individuals is increased on the average 86 per cent in women and 64 per cent in men. The total intake of oxygen is increased 100 per cent in women and 70 per cent in men, whereas the amount of oxygen absorbed by the tissues is increased to 163 per cent in women and to 95 per cent in men. The excess of the oxygen taken in by the lungs over that absorbed by the tissues is disposed of by its chemical union with the carbon to form CO_2, and the increased interchange of gases thus revealed indicates an exaggerated and abnormal waste of tissue.

In spite, therefore, of an actual reduction of respiratory capacity there is an augmented activity on the part of the lungs. This increased exchange of gases is found to exist throughout all stages of the disease, even up to a few hours before death, and lessens with return to health. According to Robin, this exaggeration in the production of carbonic acid and in the amount of oxygen both taken in and absorbed is so constant that it may form an important aid in the detection of early tuberculosis. It proves also that these patients do not suffer from oxygen starvation, as is popularly supposed, but on the contrary consume nearly twice as much oxygen as in health.

There is, consequently, not a diminution but rather an augmentation of carbonic acid produced, and hence one might expect a corresponding sense of air-hunger, i. e., dyspnœa. Such would be the case certainly were CO_2 retained in the system, as in heart disease and asphyxia; but since the intake of oxygen is increased there is no such accumulation of CO_2, and therefore the respiratory center is not abnormally stimulated, as proven by the want of marked acceleration of breathing.

On the other hand, we are confronted by the clinical observation that patients with acute miliary tuberculosis of the lungs not infrequently manifest objective and subjective dyspnœa, although in them the exchange of gases must be increased according to Robin's observations the same as in other forms of tuberculosis.

We are forced, therefore, to search for some other explanation for the lack of dyspnœa in ordinary cases of pulmonary tuberculosis than that of an alteration in the respiratory activity.

Cornet finds this explanation in the manner in which pulmonary tuberculosis develops. The striking discrepancy between the great extent of lung destruction and the slight difficulty in breathing is explicable, he thinks, only on the hypothesis that the gradual development of the disease allows the organism to adjust itself to the change. In this respect it is analogous to the state of things in pleuritic effusions. If the lung be rapidly and extensively compressed, urgent dyspnœa is experienced, whereas little or no respiratory disturbance is occasioned by an effusion that takes place insidiously. Cornet's explanation has much to commend it.

Although freedom from distressing breathlessness is the rule in chronic cases, still exceptions do now and then arise in which distressing dyspnœa is observed. As previously stated this is the result of some complication rather than of the primary disease. Of these complications pneumothorax is the most distressing and serious. If the rupture is valvelike, so as to permit the ingress but not the egress of air from the pleural sac, the pressure becomes so excessive as to plunge the patient into a condition of direst distress and directly to threaten life.

Another condition giving rise to shortness of breath is emphysema. This is not a common complication of pulmonary tuberculosis, but there are cases, either of vesicular emphysema to which tuberculosis is added or of primary tuberculosis to which compensatory emphysema becomes added. In all such cases the patients manifest breathlessness the same as in ordinary large-lunged emphysema without tuberculosis.

Tuberculous subjects with kyphoscoliosis, or any other condition which curtails the respiratory capacity of the opposite healthy lung, e. g., an effusion into the pleural cavity, are likewise apt to experience dyspnœa, particularly on exertion. The same result follows right-heart failure in cases which display very considerable abrogation of lung capacity from extensive fibrosis, although such is by no means always the case.

Exceptionally, cases are met with that manifest asthmatic paroxysms. I cannot now recall having seen such, but Cornet mentions the instance of a lady who, during several months, suffered from attacks of dyspnœa which seemed to threaten suffocation and persisted for a number of minutes.

Cough.—This is so invariable a symptom of pulmonary tuberculosis tha one cannot think of the latter without the former. It is very variable i degree and frequency and severity, however. In most cases cough is initial manifestation of the disease, although it may be so slight, a m hack, as to escape attention or be disregarded alongside of decline in we and strength. In the bronchitic form cough is more insistent and is to be very troublesome. It may still be a short, dry hack that is frequ repeated, but more often in this class of cases it is severe, prolonged and even paroxysmal.

In the incipient stage as well as throughout the whole course of the disease cough is likely to be most annoying at night; but I have known patients to declare that they lost their cough when they lay on the affected side. Sleep is prevented or is interrupted, and as the irritation occasioning the cough is not removed or not relieved for more than a few minutes at a time the cough is soon repeated. Patients are known to cough in this manner day and night, so that the symptom wears on the nerves of the friends as well as on the strength of the individual himself.

When, in the incipiency of the disease, cough is so violent, frequent and uncontrollable, it may be owing to laryngeal involvement, and hence the larynx should be carefully examined. In some instances I am sure there is a nervous element which increases the cough. The person displays that abnormal heightening of nervous irritability known as erethism and hence the bronchial irritation calls forth an exaggerated response. The patient frequently says he feels as if there were something in his chest which he cannot expel, and accordingly he makes no effort to restrain his inclination to cough. The effort may be unproductive, but it generally results in the expectoration of a scanty, clear, slightly frothy mucus.

As the malady progresses and softening occurs the symptom now under consideration changes somewhat in character. Instead of being a hard, dry, irritating cough it now becomes loose, as we say, but in this respect cases do not all agree. The cough may still be frequent and more distressing at night and in the early morning than during the day. The symptom is very apt to be excited by deeper inspiration than ordinary and hence generally comes on during a chest-examination. Some persons in this stage give only a short tussive effort and raise with ease, while others are obliged to cough for several moments before relief is obtained.

The difference probably depends upon the state of the bronchial mucosa, there being in some cases considerable inflammation with free secretions, or else upon the relation of the caseous and ulcerating focus to the air-tubes. In these latter cases the bronchi are so situated as to facilitate expectoration, while in other instances sputa are expelled with difficulty, owing to the smallness of the contiguous tube or the angle with which it communicates with the focus.

In the stage of excavation cough is still present, but it displays all possible differences in frequency and intensity. According to my observation, cough is not very troublesome after a cavity has once formed and is shut off by a limiting zone of fibrous tissue, that is, after it has become a relatively dry cavity. When, on the other hand, excavation is actively progressing or a vomica is secreting freely, cough is annoying by reason of its frequency. The consumptive is now very apt to cough upon lying down at night. This may be in consequence of the change of posture occasioning or facilitating a discharge of pus into a neighboring bronchus which still further excites the already inflamed or irritable mucosa.

If the secretions be abundant and the tube suitably located, the sputa are expelled with ease and cough is soon allayed. If, on the contrary, the bronchus be situated so high in the wall of the vomica as to be above the level of the purulent contents, or to open into the cavity at an oblique angle, the secretions are forced into the tube with difficulty and cough is necessarily hard and prolonged. In the terminal stage, moreover, the patient may be so feeble as to lack sufficient strength for effectual cough. In other instances, as death approaches, the sensibility becomes reduced and the individual seems unconscious of the accumulation in his lungs which is yet producing numerous coarse bubbling râles.

In all stages of the disease the cough is probably influenced by the degree of body-temperature. Fever seems to augment reflex irritability and hence, as pyrexia increases or comes on late in the day, cough is apt to be aggravated. The symptom is also excited by physical effort and by change of temperature, as leaving a warm room for the cooler outside air or *vice versa*. In these respects there is nothing peculiar, however, to the cough of pulmonary tuberculosis, since the same thing is seen in cases of acute and chronic bronchitis. Intercurrent complications, as dry pleurisy, are also very likely temporarily to increase the cough in frequency and severity. The same holds true of bronchial congestions, and hence patients frequently say their cough has been worse on account of their having taken cold. Doubtless such is the case at times, but often the aggravation of the symptom is owing to an advance in the tuberculous process or to involvement of the pleura or larynx. Consequently, at such times, the chest should be carefully reëxamined and the throat and larynx closely inspected.

Expectoration.—Another more or less constant feature of pulmonary tuberculosis is expectoration. As with cough, one encounters marked differences in this symptom in both character and amount. In the incipient stage sputum may be very scanty or even absent. At first it is apt to consist largely of serum and mucin so that it presents a frothy glairy appearance and is raised with difficulty. At this time patients are quite likely to declare that they have no expectoration or spit out only saliva. In this initial period the characteristic bacilli also are apt to be wanting.

In a relatively small number of cases blood is expectorated in this stage. It most often occurs as a reddish stain or streaking of the sputa, but now and then the blood may be so abundant as to merit the term hæmorrhage. In such an event it is bright and frothy (see Pulmonary Hæmorrhage), and in amount it may vary from a drachm or two to several ounces. Not infrequently it is this circumstance that first brings the individual in alarm to the physician. Occasionally the expectoration is so intimately admixed with blood as to present the appearance of raw meat (Cornet). Hæmoptysis, in the incipiency of the disease, is rarely so copious as to prove a dangerous syr tom, but during softening and excavation the blood may come from a ruptu pulmonary vessel; then the hæmorrhage may be so profuse and repeated as prove a source of peril, and even a cause of death.

With the progress of the disease the sputa assume a different chara in consequence of the secondary mixed infection. They now become purulent or even wholly purulent, passing from a grayish-yellow hue yellow and in some cases to greenish color. According to Cornet the yellow color may be imparted to the sputum by the bacillus aureus and squamosus or marcina lutea aurantiaca and variagata (Panizza); whereas the bacillus pyocyaneus imparts to the expectoration its greenish hue, this last-mentioned organism having been identified by Cornet in the interior of vomicæ. There is no doubt, however, that the expectoration may be yellowish from the presence of streptococci.

A character of the sputum to which formerly diagnostic significance was attached is that known as *nummular* or coin-shaped. It consists of solid masses which sink to the bottom of the sputum-cup and there spread out into roundish, slightly flattened globules whose outer margin is somewhat ragged or fringed and whose surface is apt to be covered with frothy serum. These nummular masses were once, and by some are still, thought to emanate from tuberculous vomicæ but are now known to come also from bronchiectatic cavities.

Differences are also observed in the ease and amount of expectoration. Some patients cough almost incessantly and yet raise little or nothing, this, according to Cornet, being the case in lymphogenic and hæmatogenic forms, while others bring up their sputa without difficulty and in relatively large amounts. Such, in my experience, is the case in the bronchitic type. With the advent of softening the expectoration grows easier and is more copious. Some may raise only a drachm or so in the early morning and then but a little more during the day. Others may expectorate even as much as half a pint of yellow purulent secretions, especially when there are old pus-secreting vomicæ.

Toward the end of the disease the sputa may diminish considerably either because the destruction of lung is not progressing, or on account of such feebleness as to render expectoration practically impossible. In the terminal weeks of life such bedridden consumptives sometimes emit from the chest fine dry clicks or crackling râles which, as Cornet says, may be audible some little distance away. Piorry, who first called attention to such sounds, explained them as produced within a cavity situated at an apex and propagated to the external world through a large freely communicating bronchus. I have often noted this phenomenon but did not understand its mode of production.

As regards the various components of the tuberculous sputum the greatest importance is unquestionably to be attached to tubercle bacilli. These vary enormously in number and frequency of appearance but probably never fail to be present at some time or other in the course of the affection. They are shown in Plate VII. By *closed tuberculosis* is designated cases in which bacilli are wanting and by *open tuberculosis,* those in which bacilli are present in the sputum.

Several conditions may account for the absence of bacilli. In miliary tuberculosis, or in any other form so long as the tubercular foci do not ulcerate and are not discharged through a bronchus, or when a caseous zone fails to communicate with an air-tube, bacilli are wanting. Hence, early in the pulmonary process one may search repeatedly for the germs before any are discovered. On the other hand, the breaking down of a focus and its evacuation through a bronchus may be the reason why bacilli are sometimes found in immense numbers. The former circumstance explains why it is not wise to measure the prognosis by the absence of bacilli. The process may be widespread and yet no germs be found for weeks. On the other hand, the detection of vast numbers of bacilli although usually taken to indicate a rapidly ulcerating process does not necessarily betoken a bad outlook, since with the elimination of the focus the disease may come to an arrest. For this reason the gradual disappearance of bacilli is generally regarded as a favorable indication.

During the stage of ulceration the sputum also contains elastic fibers, and when the destructive process is extensive even entire fragments of pulmonary parenchyma. Fraenkel is of the opinion that this latter is only apt to take place when a zone that has been inclosed and shut off by fibrous tissue undergoes ulceration. It is likely to occur, therefore, when such a chronic tuberculous or fibroid lung becomes gangrenous. Elastic fibers may sometimes be readily distinguished by the naked eye. At other times the sputa must be treated with strong acetic acid or a dilute solution of potassium or sodium hydrate and then centrifuged and examined microscopically.

When mixed infection has supervened other bacterial forms are discovered, either to the exclusion of bacilli or in combination therewith. For the most part these are streptococcus pyogenes, but staphylococcus, bacillus coli communis, pneumococcus and bacillus pyocyaneus (Cornet) have also been identified by Petruschky and many others. Doubtless many of the streptococci are from the mouth and upper portions of the air-tubes, but bacteriological investigations after death have proven beyond peradventure the presence of pyogenic bacteria in the lungs in intimate union with tubercle bacilli. Such pus germs have also been obtained in almost pure cultures from the purulent contents of vomicæ. It was in such cavities that Cornet discovered the bacillus pyocyaneus, a rare circumstance certainly, since Kerschensteiner failed to identify this germ in his cases.

Various other not very important constituents of the sputum, although many foreign observers have spent much time in the discovery and estimation of these ingredients, are the following: water, 94 to 95 per cent; inorganic salts as phosphates and sodium chloride, organic matters, as mucin, extractives, albumin and fat (Renk), epithelium cells from the air-passages, pigment, granules, myelin, alveolar cells, detritus, leucin, lecithin and cholesterin (Bokay), tyrosin and coral threads (Leyden), glycogen (Salomon), urea (Fleischer) and nuclein (Kossel).

Circulatory System.—The *pulse* is accelerated and of low tension even in the incipient stage of pulmonary tuberculosis, but exhibits considerable variation in these respects. Individuals who present themselves for a medical opinion regarding the state of their lungs are very apt to be nervous and apprehensive and to show a frequent pulse on this account. But when any such influence can be excluded the heart's rate will almost invariably be much too rapid to correspond with the temperature. Accordingly, the pulse may range anywhere from 90 to 120.

Indeed, so constant a feature of incipient tuberculosis is increased frequency together with lowness of tension about to be mentioned that in doubtful cases it may aid one in arriving at a diagnosis. I have many times found the pulse-rate below 90 but it is certainly very rare to find it as low as 80. I recall one instance, however, in which a man consulted me regarding his lungs whose pulse was slightly below normal and of such excellent tension that I was led to remark, " Well, sir, with such a pulse as this I should be greatly surprised to find anything wrong with your lungs." As the result of examination showed, my conclusion was altogether too hasty, for there was pronounced evidence of tuberculous infiltration of his right upper lobe. I am at a loss to explain the pulse-rate in this case, but as he was operated upon for an appendicitis not long after, there may have been some connection between the two.

Lowness of tension is another feature of even greater import than its frequency in tuberculous subjects. This is commented on by most writers and has been amply proven by the sphygmograph and sphygmomanometer. That the pulse should be weak and soft when running at 120 or 125 is not strange, but even when its rate is not over 90 it will often feel weak to the finger.

That the above-mentioned characteristics are not solely or mainly owing to anæmia and debility is proven by the blood-state. The individual may look anæmic and may, in some cases, have the so-called pseudo-chlorosis, but such is by no means always the rule. Neither is the degree of fever responsible for the frequency and feebleness of the pulse, since in incipient cases the temperature is not high. The explanation is probably that given by Cornet, namely, the tuberculous infection itself. The evolution of tubercles is attended by the formation of toxins and it is their absorption which probably accounts for the low blood-pressure, as well as for other initial symptoms. Of course, considerable variation in this regard is observed in different cases and this depends on differences in degree of the toxæmia.

Later in the course of the disease, increased frequency and feebleness of the pulse are to be expected. There are several conditions likely to bring this about. The patient is weak and emaciated, his digestive processes are defective and he is sadly undernourished. He is poisoned, moreover, by the toxins of mixed infection and often shows hectic fever. Under such circumstances it would be strange if he did not exhibit a rapid and feeble pulse, but when all these influences are given due allowance it will still be seen that they do not wholly explain the peculiarities of the pulse.

Even during the hours of the day when pyrexia is absent the pulse-rate remains high, while after the consumptive has begun to mend, as shown by gain in strength and weight, his blood-pressure remains low. There is now, as in the incipiency of the disease, a degree of cardiac debility only explicable on the hypothesis of a profound infection, in other words a toxæmia for which the tubercle bacilli alone are responsible.

The Heart.—But little need be said concerning this organ. We know by morbid-anatomical researches that tubercles may invade the myocardium and the pericardium and, in the opinion of some (Teissier), the bacilli may even produce endocarditis. Clinically, however, it is among the rarities of medical practice to recognize such lesions.

Upon physical examination the heart is generally found practically unchanged. The sounds may be weak or at the apex there may be a faint systolic whiff, which is either an accidental murmur or at the most may indicate a muscular insufficiency of the mitral leaflets. The pulmonic second tone is apt to be accentuated, but even this is not invariable. The aortic second sound is quite likely to be more or less diminished. In all these, however, there is nothing more than what can be explained by simple cardiac debility. In some cases, when the lungs are extensively destroyed, there may be signs of right ventricle dilatation, but even this is often far less than what might be anticipated.

In a few instances I have seen a degree of cardiac feebleness and excitability that led me to suspect myocardial or pericardial involvement, but, although I systematically auscultate for evidence of pericarditis, I have never yet detected a positive friction murmur. In only one instance have I run across a distinct mitral regurgitation, and in that case there was history of acute articular rheumatism prior to the outbreak of pulmonary tuberculosis. I believe the association was purely accidental and that it was an instance of the development of pulmonary tuberculosis, despite the existence of a previous mitral valve lesion. This case was narrated in my work, "Diseases of the Heart and Arterial System."

In this connection it may be well to mention briefly the interesting case of Miss N. This young lady was a victim of pulmonary tuberculosis of one apex for which she was sent to Colorado. There she remained through the Winter and by June had so far improved that she returned to Chicago for a visit. Against my emphatic warning she remained at home until late in the following Autumn.

Then, as the result of a cold, as she fancied, this young woman suddenly experienced an aggravation of her symptoms and was found to have incipient involvement of her other apex. She was hurried off to Colorado where she improved for a second time. She did not regain her previous excellent state of health, although her pulmonary signs became quiescent.

At length some two years subsequent to her second return to Denver this patient died suddenly. Her symptoms were stated to have been chiefly signs of cardiac weakness. The *post-mortem* examination revealed complete arrest of the tuberculous process in the lungs, but these were extensively fibrosed. The discovery of chief interest was in the heart. Both ventricles were thin-walled and the right ventricle was demonstrably dilated because its wall had become as thin as paper, to quote the remark of a physician who witnessed the autopsy. There was no other microscopic evidence of myocardial disease.

The Blood.—There have been numerous investigations into the state of the blood in pulmonary tuberculosis. They are too elaborate and too many to be reviewed at length in a work of this kind, and therefore the attempt will be made to state in a general way the results which are to be accepted as established.

Hæmoglobin.—This component of the blood has been studied extensively both chemically and by means of the spectroscope. Whereas it is constantly reduced in tuberculosis of the bones, the investigations of Leichtenstern, Gnezda, Barbacci and Oppenheimer showed there were cases of pulmonary tuberculosis in which the hæmoglobin percentage was above normal. On the other hand, a decrease was discovered in all cases by Quinquaud, Wiskeman, Laache, Neubert, Swan and Ullom; many others have found a moderate reduction of this constituent in the early stage. In second-stage cases on the contrary, it is normal or slightly decreased (Limbeck, Appelbaum, Ewing). In the cases of the third stage it is quite uniformly reported as reduced. It may be stated, therefore, that the normal finding in the second stage is due to a concentration of the blood, so that absolutely the hæmoglobin is probably diminished.

Von Moraczewski reported a diminution in the hæmoglobin without a corresponding decrease in the iron, a reduction of the serum-albumin with an increase of the potassium salts and the appearance of cellulose which L. Freund had discovered in the blood of tuberculous subjects (Fraenkel). The fibrin is increased in the first and second stages and decreased in the third; during the course of the disease the iron, the phosphates and the salts of potassium are progressively reduced, while the sodium sàlts are increased. He agrees with Grawitz in the relative thickening of the blood in the second stage.

Erythrocytes.—The reports concerning the number of the red blood-cells vary somewhat, and yet it may be stated that in general the observers are agreed in finding them reduced in the incipient, normal or increased in the moderately advanced, and decreased in the advanced cases. The reduction in the number of the erythrocytes in early cases is not proportionate to the diminution of the hæmoglobin, however, a condition to which the term *pseudo-chlorosis tuberculosa* has been given. This similarity to chlorosis was first pointed out by Soerenson but is now quite generally recognized. It may, in doubtful cases of latent and incipient tuberculosis, prove of diagnostic aid.

The relative increase in the number of the red cells in the second stage has been considered due to a relative concentration of the blood produced by the sweating, the profuse expectoration, diarrhœa and perhaps other factors. Ullom and Craik, in their recent and exhaustive review of this interesting subject, cite the explanation given by Gaertner and Roemer to account for this increase in the erythrocytes as follows: the experiments of these investigators show that the extracts of the tubercle bacillus produce an alteration in the relations between the blood-vessels and the tissues in the interchange of lymph, with an excess in favor of the tissues. There results, consequently, a relative thickening of the blood, in accordance with the belief of earlier observers, only the explanation adduced by them was incorrect.

In the stage of excavation the noticeable reduction of the erythrocytes finds explanation in part in the general intoxication which occasions the phenomena of hectic, for cases with high fever show a more marked decrease than do others. In exceptional instances the reds may be enormously reduced as in a case of Melassez in which they numbered only 980,000. The influence of hæmoptysis may be very marked in causing such a reduction.

Ullom states that the erythrocytes have not received especially searching study as regards microscopic changes in these cells, but it may be stated that anisocytosis, poikilocytosis and nucleated reds are seldom found, the last being very rare even after hæmorrhage. Maragliano believes that a polychromatophilia has an unfavorable significance as regards prognosis even when otherwise the cases appear favorable.

As respects the bearing of the red-cell percentage upon prognosis, it may be stated that whereas their great reduction is a grave omen, their normal or increased percentage cannot be taken to denote the opposite, since the hæmoglobin and erythrocytes are said to have been found increased in cases that were developing general tuberculosis and hastening toward a fatal termination (Ullom).

Leucocytes.—Most observers are agreed in the statement that the number of the white cells is generally more or less increased. This is true, at all events, of cases presenting evidence of mixed infection and therefore in the second and third stages. In pure tuberculosis Pick, Rieder, Warthin, von Jaksch, Galbraith and numerous others (Ullom) have found that the leucocyte counts were always either normal or below. This accords with my observations which, although not numerous enough to be of scientific value, have yet led me to look upon latent and incipient pulmonary tuberculosis as likely to display a tendency toward leucopæmia rather than hyperleucocytosis.

Ullom's counts agree with those of previous investigators in showing a moderate hyperleucocytosis in cases which have passed the stage of pure and unmixed tuberculosis. His counts ranged from 5,000 to, in a single instance, 20,000. He agrees with Ewing in his conclusions that when there is an increase in the number of the white cells, it indicates or rather depends upon a suppurating cavity, an advancing pneumonia, severe anæmia or hæmoptysis.

The character of the leucocytes as well as their number has been the object of many studies. There is much diverseness of results in consequence, as Ullom says, of different groupings of the cases studied and of disagreement in classification of the cells. Einhorn and Neubert found the polymorphonuclears were in excess in the various stages, and recent observers seem to coincide in their findings as regards hyperleucocytosis. Appelbaum noted an increase in the transitional forms accompanying that in the polymorphonuclears. On the other hand, Ewing and Halbron in acute and most cases of chronic tuberculosis found the large mononuclear cells in excess, while Galbraith noted such an excess under the influence of a diet rich in proteids. Strauss and Rohnstein were inclined to believe that with progress of the disease the mononuclear forms displayed a tendency to decline.

As to the eosinophiles it may be stated that Cabot does not attach much significance to either their presence or their absence. All other observers regard them as of some prognostic importance. They were decreased in febrile cases according to Zappert, and increased either subsequent to, or directly preceding, the disappearance of fever in occasional instances. Holmes, Appelbaum, Warthin and Swan noted that in severe cases the eosinophiles were either absent or existed in only small numbers. On the other hand, various observers (Claude, Zaky, Appelbaum, Galbraith) noted their presence in early cases or, if at first absent, that they reappeared during treatment. Halbron looked upon them as an indication of the resistance of the organism to infection and Teichmueller held them of favorable significance when they appeared in the sputa.

Ullom's conclusions are as follows: (1) In pulmonary tuberculosis without cavity formation a mild anæmia with a decrease in erythrocytes and a relatively greater decrease in hæmoglobin is constant. (2) From the standpoint of prognosis an increase of the erythrocytes in cases without cavity formation is of favorable significance. (3) In advanced cases a decrease of the leucocytes is of unfavorable import. (4) In our investigations we have received the impression that the actual increase of lymphocytes seems to correspond to the increase of resistance on the part of the organism to the tuberculous infection, but further study is required to confirm this deduction. (5) The transitionals seem to follow the same rule as the lymphocytes in this regard. (6) At the beginning of the investigation the eosinophiles seemed to increase with the patient's improvement, but further study did not support this view.

A very interesting and unusual contribution to the study of the blood in pulmonary tuberculosis has recently appeared from Leube's laboratory, and is from the pen of Joseph Arneth who has published previous papers on the behavior of the leucocytes in infectious diseases. In this particular work Arneth has departed from the investigations of others in that he confined his attention to the neutrophile cells. The following is a concise statement of his results.

Arneth divides the neutrophiles into five classes according to the number of their nuclei and again subdivides each group according to the shape of the nuclei whether round, hollowed out or looplike. Thus, Group 1 comprises cells with 1 nucleus and is subdivided into myelocytes, cells with nuclei but slightly hollowed out and cells with nuclei deeply hollowed out. Group 2, neutrophiles with 2 nuclei, has 3 subdivisions, viz., cells with 2 round nuclei, cells with 2 looplike nuclei and cells with 1 round and 1 looplike nucleus. Group 3 has cells with 3 nuclei which are subdivided into 4 varieties. Group 4 is made up of neutrophiles with 4 nuclei, subdivided into 5 subordinate groups. Group 5 includes cells with 5 or more nuclei, and is subdivided into 5 subgroups.

Without entering into a detailed enumeration of the normal percentages of the five groups with their subdivisions it may be stated in general that, in normal neutrophile counts, the largest percentages fall in groups 2 and 3, whereas in tuberculosis, as in other infections, there is a tendency for the neutrophile leucocytes to belong very largely to the first and second groups. The groups with 3, 4 and 5 or more nuclei, which Arneth regards as older forms, sink into insignificant percentages. This abnormal distribution of the neutrophile cells which tends to shut out these older polynuclear elements is regarded by Arneth as an evidence of defective resistance on the part of the organism, and an index of the intensity of the infection.

This conclusion seems justified by the observation that whenever the individual showed a tendency to return of the neutrophiles to their normal relative percentages there was a corresponding improvement in his condition. On the contrary, whenever the individual appeared to be improving in his general health, but the abnormal arrangement of the neutrophiles persisted, it invariably betokened a bad prognosis, for the local disease was sure to reassert itself in the course of time.

Moreover, although in cases of mixed infection there might be a hyperleucocytosis, this was usually moderate and the preponderance of the cells with one and two nuclei still persisted. In short, he believes that in the characters of the neutrophile leucocytes is to be found a measure of the intensity of the infection and of the organism's ability to resist. Finally, under a successful tuberculin treatment, Arneth finds that the neutrophile cells approach the normal and that in case of pronounced reaction they again become morphologically and relatively altered, as in any infection.

Digestive System.—The majority of consumptives have more or less disturbance of appetite and digestion, but in this regard striking differences are encountered. As previously stated there is a class of tuberculous individuals whose malady appears to set in with dyspeptic symptoms and on this account they fancy their trouble is entirely gastric. They may or may not display anorexia, though they more commonly do, and in addition complain of weight or pain in the epigastrium, eructations or a feeling of fullness after eating, nausea or even vomiting. Such cases are most trying to treat and are apt to furnish a bad prognosis.

The most usual complaint is of want of appetite or, if desire for food is retained, of inability to take the proper amount because so quickly satisfied. Some of these patients are able to force down simple articles of diet that do not offend the palate or the eye, while others can consume only such dainties as actually appeal to their gustatory sense, displaying disgust or repugnance for plainer and more wholesome fare. Still others have such an unconquerable anorexia as to be unable to swallow even the most attractive articles of food. Their anorexia is so complete and many times they are so unwilling even to attempt to eat, that they exhaust the patience of both doctor and friends.

In a smaller number of individuals the appetite is unimpaired or is even so great as to constitute a bulimia. They declare they have no trouble with their stomachs and their evident ability to eat and digest whatever is set before them affords a refreshing exception to the opposite class. It is generally found that such persons have always had a fine appetite and that they take large quantities of food from relish. The prognosis in such cases is, on this account, apt to be relatively good and one sometimes wonders why they did not maintain such a state of nutrition as would have protected them from tuberculosis, that is, enabled them to resist the infection in its beginning. In the same way one wonders why some consumptives improve so rapidly as soon as their ability to take food is restored and why others with apparently undiminished appetite and digestion go on losing weight and strength in spite of the amount of nourishment consumed.

The cause for these differences, as well as for the intractable anorexia or gastric disorders mentioned, is probably to be sought in the toxæmia, since in the early stage there is not fever enough to account for these disturbances. When, in confirmed phthisis, there are the symptoms of mixed infection, it is not difficult to understand why appetite and digestive capacity may be lost. Cornet attributes the gastric derangements to absorption of "proteins" from the lungs, while Fraenkel thinks they may be referred to a primary disorder of the nervous system, either a local manifestation of a general neurasthenia or an effect of toxins on the nervous apparatus. He would also attribute the digestive symptoms in part to the anæmia so often seen in tuberculous persons. Gueneau de Mussy and Peter suggest that in some of these cases there is pressure upon the vagus by enlarged lymph nodes and have reported such an instance, but, as stated by Cornet, there should also be in such a case tachycardia and an irritative cough.

A host of investigators has endeavored to throw light on this subject by examinations into the motor function of the stomach and the chemical constitution of the gastric juice. Their findings are contradictory and prove conclusively that no constant rule obtains. Thus, Brieger found normal gastric function in 50 per cent of incipient cases, in 33 per cent of moderately severe cases and 16 per cent in severe cases, while in the balance of his cases there was more or less pronounced gastric insufficiency of high degree (Cornet). On the contrary G. Klemperer in nearly all cases of the initial stage found more or less motor weakness, while for the most part there was a hyperchlorhydria (Fraenkel).

Other observers have found a diminution in the motility and acidity especially in the later stages of the disease. Hildebrand demonstrated free hydrochloric acid as long as the temperature remained about normal, whereas it was absent in continued fever, invariably when the pyrexia reached 101° F. This finding by Hildebrand was first cited to me by the late Dr. Dettweiler in 1893, who made it a rule to reduce the temperature in such cases, by small doses of antipyretics, as suggested by Hildebrand, before administering food. Dettweiler claimed that by such means he could get his febrile patients to digest considerably more nourishment than otherwise.

Vomiting is not uncommon in chronic phthisis and when accompanied by epigastric pain and distention may be owing to gastric catarrh or, as is more frequent, to reflex influence from cough. In this latter form, the vomiting is very apt to take place soon after a meal and follows directly upon a coughing spell. Wilson Fox says such reflex emesis is more common in chronic pulmonary tuberculosis than in any other lung disease save pertussis. This type of vomiting is not associated with nausea or epigastric distress and occurs with great suddenness. It may be so frequent as seriously to interfere with the patient's nutrition.

Intestinal symptoms are not rare in consumption and are varied. The most frequent disorder is constipation which, by Cornet, is ascribed to hyperacidity. It may be very obstinate or may alternate with attacks of diarrhœa.

It is associated in many cases with annoying flatulence in which event there may be slight griping pains. It is also worthy of note that the exhibition of a mild laxative in these subjects may sometimes exert a far more powerful effect than was anticipated. A man with chronic ulcerative phthisis of the right lung recently declared to me that a tablespoonful of castor-oil taken in orange juice caused him excruciating colic. He had four small evacuations after which his cramps were so severe that for two hours he lay on the floor doubled up with agony. This man presents no symptoms suggestive of intestinal tuberculosis, but inasmuch as Traube maintained that ulceration of the bowel might exist without diarrhœa (Fox) it is possible that in this instance such was the occasion of his violent cramps.

Diarrhœa is a far more serious symptom and one that may be so intractable as to give the practitioner much uneasiness. It is looked upon with dread by both laity and many physicians as indicating intestinal tuberculosis. Such is, however, by no means its sole or even its most usual cause, but to this I shall return anon. Looseness of the bowel is very apt to set in after a chill from exposure or some indiscretion in diet or may come on from no assignable cause after a period of constipation.

Diarrhœa is generally a late manifestation in the course of chronic pulmonary tuberculosis, but now and then cases are seen in which it was an early symptom, so troublesome as to have masked the lung mischief for a time (Cornet, Fox). There may be no obvious *post-mortem* findings to account for this symptom, and it is presumable that an intestinal catarrh existed, as suggested by Fox, or there may be some of the changes discovered by Girode and by Cornet which were referred to the action of toxins, "proteins" as Cornet calls them, on the intestinal mucosa.

The changes described by Girode and found in nine out of twenty-one cases examined by him affect the glands of Lieberkühn, on the one hand, causing these to resemble an adenoma, and on the other, the lymphatic vessels. There appears to be a lymphangitis which has led to thickening and obliteration of the lymph vessels situated between the two muscle layers, and in the main stems to stasis in consequence of obstruction by a fine granular mass resembling a white thrombus. This last condition Cornet regards as very significant and interesting since it might easily have accounted for a pouring out of serum into the lumen of the intestines and so to diarrhœa. Cornet holds also that the symptom produced by the proteins may depend upon irritation of the nerve-endings of the sympathetic, which irritation produces a reflex increase of peristalsis, or that it may be the expression of a catarrh due to the intense action of the toxins on the mucosa or of actual superficial erosions of the mucous membrane.

This illustrious author is impelled to these conclusions from analogy with what takes place in the air-tubes. Their mucous membrane is irritated and at length inflamed by the frequent passage over it of sputa containing bacteria, i. e., bacilli and pus-cocci with their toxins, even without lodgment of the germs on the mucosa. In the same manner, when phthisical sputa are swallowed, as is the case in children, insane persons and others who are too feeble to expectorate, bacilli may not be deposited on the gastric or intestinal mucosa because of the dilution and mixture of the sputa by food and fluids, yet may irritate and inflame it in the passage over it which permits absorption of toxins.

Most writers take a different view from Cornet and regard the intestinal catarrh and ulceration as a result of the action of the bacilli themselves. That a veritable tuberculosis of the intestinal tract can and does take place is amply proven by *post-mortem* investigations. However, this is, particularly in the chronic form of pulmonary phthisis, a secondary complication and not a primary condition.

Indeed, it is well established that a primary tuberculosis of the bowel is exceedingly rare in adults. It may occur in infants fed on contaminated cow's milk, but in grown persons it is so infinitely rare that Fagge is said by Fox to have maintained that such never occurs. Cornet also holds to its extreme rarity and cites Eisenhardt's statistics in proof of this position. Of 1,000 consumptives examined *post mortem* in the Pathological Institute at Munich there were 566 instances of secondary intestinal tuberculosis, i. e., 56.6 per cent, and only one of primary disease of the bowel.

Consequently, *one should be very cautious* in making a diagnosis of tuberculous invasion of the intestinal tract when diarrhœa comes on in the beginning of the lung disease, or when the sputum is known not to have been swallowed, *unless one can at the same time determine general tuberculosis.* Especially should one hesitate to declare an adult to be suffering from this intestinal involvement when the lungs are not also affected. I have many times been told that an individual had died of tuberculosis or, as the laity says, consumption of the bowel, yet without disease of the lungs, and I have always believed there was some error, i. e., either the diarrhœa was not of a tuberculous origin or the lungs were also and primarily involved, yet without having been recognized as such.

But to return! Secondary intestinal tuberculosis is induced by careless or voluntary swallowing of the sputa. Bacilli are in this manner carried into the stomach and not being there destroyed are passed on into the small bowel, whence they in time reach the colon.

Even if ulceration does not result, a catarrh is very likely to ensue and an obstinate and exhausting diarrhœa to become a prominent symptom. For this reason consumptives should be carefully warned against this habit. It is, moreover, a dirty trick that well-nigh turns one's stomach at the thought. Unfortunately children and a few others cannot be prevented from it and hence it is that they and feeble-minded or insane patients so often display combined pulmonary and intestinal consumption.

There is nothing in the stool so characteristic of intestinal tuberculosis as to make the diagnosis certain. Wilson Fox speaks of the discharge as light yellow, pultaceous and often frothy, while Fraenkel says that profuse thin brownish passages are occasioned by deep-seated ulceration of the large bowel.

Pain is generally a concurrent feature, the patient experiencing a cramp-like pain immediately before stool and being tender to pressure over the transverse colon. Such symptoms are not invariable, however, nor always reliable for intestinal ulceration may exist without any clinical manifestations whatever. It may be put down as true in at least half of the cases of chronic phthisis that when persistent diarrhœa sets in toward the end of the malady it is probably owing to tuberculosis of the bowel.

The Skin.—Perspiration is one of the most distressing symptoms in cases of chronic pulmonary tuberculosis. It varies much in the date of its appearance as regards the course of the affection and in its severity. In my experience this feature of the malady is not a subject of much complaint in incipient cases. There may be a tendency to break into a perspiration with rather less provocation than is the rule with healthy persons, but the patients do not dwell on it in particular.

Now and then however an individual with incipient phthisis is encountered who makes especial mention of sweating at night. In some cases the symptom is undoubtedly annoying enough to attract attention and occasion considerable discomfort, but in others I am sure that the occurrence of perspiration is mentioned because, in the mind of the laity, it is associated with consumption and hence, even when slight, is calculated to frighten the patient and his friends.

Later in the course of the affection when to the tuberculous has been added a streptococcus infection sweating becomes distressing and even so profuse as to be weakening. As the perspirations occur most abundantly at night they are spoken of as *night sweats* and are apt to be regarded with apprehension as belonging to the last stages of consumption. The patient is waked out of sleep somewhere after midnight to find his body bathed in cold perspiration and his night clothes so wet and uncomfortable as to necessitate changing. Dried by his attendant and put into warm dry garments he again falls asleep, and on awaking in the early morning hours is likely to be once more in a profuse sweat.

Such drenching perspirations, termed colliquative, are truly exhausting, and in several instances coming under my observation formed a scarcely less fruitful source of complaint than did the cough. In advanced cases of chronic phthisis they are most difficult and perplexing to treat and I have known them to defy all vaunted remedies. Although quite general in most cases the sweating may yet be more marked about the head and upper part of the trunk, the sufferers not seldom finding their hair thoroughly soaked.

Why such is the case I cannot explain, nor do I recall a satisfactory explanation. The perspiration itself is probably a manifestation of toxæmia, being due, in the early stage, to the toxines of the bacilli and in *phthisis confirmata*, to the mixed infection. Other reasons have been assigned for the occurrence of the perspiration of this disease, particularly in cases that do not display much if any toxæmia. The only one, however, that appeals to me is that of the feebleness of the circulation.

I am inclined to this view by my observation of profuse and irremediable sweating in some cases of grave cardiac inadequacy, notably two in which the arterial stream was remarkably weak and sluggish from aortic stenosis. In one in particular there was no elevation of temperature, but rather a depression, and no increase in the leucocytes indicative of infection. The perspirations were present by day but were especially severe by night, or whenever the man awoke out of sleep, even by day. They grew less only as the circulation improved after weeks of rest in bed and other appropriate measures.

Doubtless perspiration is influenced in many cases not tuberculous, by the state of the vaso-motor system, that is, is brought about through the action of the nervous system, as during nausea, fright, etc. In incipient tuberculosis, cardiac disease and great weakness, exertion, etc., the action of the vasomotor centers on the sweat glands cannot be ignored; but whatever be the exact mode of its production I do not believe it can be attributed solely to the action of toxins. Definite views on this subject are certainly desirable from a therapeutic standpoint if from no other.

Before leaving this subject of the perspiration I desire to mention the very disagreeable, penetrating odor which I have often noticed about chronic consumptives. It is peculiar and indescribable, and different from that perceptible in cases of pus infection. It has no dependence, moreover, upon the patient's habits as to bathing, i. e., is not due to untidiness of person. This odor is so distinctive that my nose has often enabled me to make a diagnosis before resorting to a chest examination.

Desquamation of the skin is not at all uncommon in phthisical patients and when most marked is caused by pityriasis versicolor. This affection of the integument is owing to the presence of a vegetable parasite and is not peculiar to consumption.

Œdema.—This symptom is sometimes seen in consumptives who are far advanced in the disease. It is justly regarded by the laity as an indication that the end is not far distant. This condition is not generally very extensive but is usually confined to the feet and ankles. It is a manifestation of feebleness of circulation which is enhanced by a hydræmia. In some instances it may be a symptom of amyloid degeneration of the viscera, in particular of the kidneys.

Venous thrombosis is another occasional cause of œdema. This coagulation of blood in the veins is probably due to a variety of causes which may act independently or in conjunction. Fraenkel attributes it to the action of toxins on the epithelium of the blood-vessels in consequence of which the coagulation of the blood is favored.

Cornet, on the other hand, thinks it due in some cases to feebleness of blood-flow intensified by prolonged decubitus or now and then to pressure. The fact of venous thrombosis in a variety of other infections, as typhoid fever, makes it probable that the action of toxins is its chief if not its sole cause. This view is also supported by the circumstance of its occurrence in early as well as in late stages of the disease, although this latter period of its development is the more usual.

Thrombosis is not a frequent complication as shown by statistics given by Fraenkel. Thus, out of 1,778 cases of pulmonary tuberculosis observed in Gerhardt's clinic Ruge and Hierokles found it but 19 times or, roughly speaking, about 1 per cent, while Dodwell saw 20 instances among 1,300 tuberculous cases, or 1.46 per cent. The only case I have seen in my private practice occurred in a man suffering from confluent tuberculous pneumonia of almost the entire left lung. The thrombosis affected the left femoral vein, as I now recall, and was early in the course of his disease.

Any of the veins may become thrombosed, but those of the lower extremities are the most frequently affected. The thrombosis may involve the popliteal, posterior tibial, or femoral veins, the inferior vena cava, the vena innominata or even a cerebral sinus. In the majority of cases a collateral circulation is established and the results of the coagulation are unpleasant for the time being, rather than permanently serious.

Nervous System.—The symptoms on the side of the nervous system which are observed in the course of chronic pulmonary tuberculosis may be conveniently considered under two heads, (*a*) psychical, (*b*) organic. I have seen occasional instances of both kinds of disturbances, but as my experience with these features of the disease has been limited I shall not hesitate to draw freely from authors at my command.

(*a*) *Psychical.*—The one peculiarity of the consumptive which probably strikes the observer most forcibly is his hopefulness, the *spes phthisicorum* of the ancients. This is not usually seen, or at least is not pronounced, in the beginning of the disease, but late in its course, when it is only too apparent to his friends that death is not far off, the consumptive is possessed with a belief in his speedy recovery. He not only talks hopefully of his condition, but actually makes plans for the future which to his friends are absurd and distressing. It is this sanguine expectancy which makes the bedridden consumptive so ready to undertake journeys to some vaunted resort.

In the incipient stage, individuals are apt to be easily discouraged and despondent, and it is on this account that the physician is usually requested not to say anything discouraging to the patient. It has generally been my observation, however, that this state of despondency is not persistent but soon gives way to one of hopefulness, especially if the information concerning his disease be imparted in a manner to arouse a feeling of cheerfulness. I have never seen this despondency so marked in any person as in a young man with tuberculosis whom I once examined in Minneapolis. Nothing would cheer him up and as a matter of fact he died of his malady not many weeks thereafter. He was advised to go to Colorado and did so, but failed so rapidly and progressively after arriving in Denver that he was soon sent home in his coffin. Ever since I have regarded such extreme hopelessness as furnishing a very bad prognosis. Such patients are very introspective and apprehensive and this frame of mind militates against recovery in any organic disease.

Other psychical manifestations have been described by Heinzelmann, and both Cornet and Fraenkel recognize the two groups into which he divides these symptoms. In the one group there is a certain irritable weakness of the psychical nature which shows itself by an inconstancy of mood, desire for sympathy, general irritability and a degree of excitability that may even prevent sleep.

In the second group there is a loss of mental balance which declares itself by the too optimistic view of his own condition and a lack of ability critically to appreciate his symptoms. In addition there is deficient energy or an opposite state of things, i. e., an inclination to undertakings for which his strength is not adequate. The patient having lost his power of persistent application, acts are decided on impulsively, and in a few cases there is an inclination to sexual excess or at least unusual venereal desire.

Cornet depicts the anxious solicitude with which a consumptive will study his various symptoms, viz., the changes in the amount and character of his sputum, his temperature, the appearance of blood in his expectoration, etc., the evident eagerness with which he watches for and seeks words of cheer, and his anger or tears over statements that seem to him gloomy. In one case he observed a temporary melancholia which was shown by an inclination to suicide.

F. Wolff is said by Fraenkel to look upon these various psychical and mental disturbances as not peculiar to the disease of which the consumptive suffers but merely as an exaggeration of his natural characteristics. With such a view I am in hearty accord, for I have seen similar and fully as pronounced disorders on the side of the emotions and intelligence in persons seriously ill from other maladies than tuberculosis. In particular, I have in mind a physician mortally stricken with cardio-vascular and renal disease whose hopefulness, tearful despondency and general instability of mood, whose plans for the future, and inability for sustained mental effort were as remarkable and incomprehensible as they could possibly be in a consumptive.

In this man they were clearly but exaggerated manifestations of his mental and emotional constitution. I do not know how an alienist might explain them, but to me they seem to indicate that under the effect of disease and physical weakness, the consumptive loses control of himself and yields to impulses which in health he would be able to recognize as irrational or excessive and hence would control.

Fraenkel says the general weakness and ready exhaustibility of these tuberculous patients can increase until it amounts to a positive neurasthenia, and Wilson Fox includes among the complications hysteria, which, he says, may increase the difficulties of diagnosis and treatment. There are not hysterical convulsions but the cough may be rendered spasmodic and there may be hysterical aphonia. Such facts serve to emphasize what was said above; namely, that these persons are often neurotic by nature, and that the pulmonary disease enhances or brings out their neuroses. It is such considerations, I suppose, which have led some writers to espouse the absurd theory that consumption is but a part or local manifestation of a disorder of the nervous system.

Delirium is another mental disturbance that may occasion great uneasiness in the mind of the practitioner. It occurs toward the end of the disease and, especially if associated or preceded by headache, may give rise to the suspicion of meningeal tuberculosis. Although, as intimated by Cornet, it may be a symptom of tubercle within the cranial cavity it is in most cases a result of increasing exhaustion (Fraenkel). Very exceptionally maniacal outbreaks may occur.

(*b*) *Organic.*—Under this head may first be mentioned various disorders of sensation: neuralgias, hyperæsthesia, and anæsthesia which are independent of neuritis and are the effect of deficient nutrition, i. e., of anæmia. The most common seat of neuralgia is in the chest and since these intercostal pains are sometimes lancinating and interfere with inspiration they are apt to be mistaken for the stitch of dry pleurisy. At other times the muscles of the chest are sore and lame so that the pain is of the character of a myalgia. Such painful sensations may be seen in early cases when the individuals are especially anæmic, but for the most part they are met with late in the affection when patients are much reduced.

Neuritis, most commonly peripheral, is a late manifestation of chronic phthisis and affects the extensors of the lower extremities more often than the upper. In the few instances that have come under my notice the legs were affected and there was foot-drop. The first case I now recall was in a young woman in whom the neuralgic pains in the lower extremities were marked. She was bedridden and in the last stages of consumption, with hectic fever and extensive destruction of both lungs.

The changes that take place in the nerves have been the subject of much study in Germany and France, and valuable contributions have been made by Joffroi, Eisenlohr, Strümpell, Oppenheim, Vierordt, Heine, Pitres, Vaillard and others. The last two authors distinguish three classes of cases as follows:

(1) Those in which, clinically, the neuritis is shown only by neuralgias that may be widespread but are transient, yet in which serious histological changes are discoverable *post mortem*.

(2) Amyotrophic neuritis in which the prominent feature is isolated or diffuse muscular atrophy and which may develop within six days from the onset. When diffuse the paralysis may be general and involve even the diaphragm. Nevertheless, certain groups of muscles may escape and thus permit limited motion.

(3) That form of neuritis in which disorders of sensation are the chief symptoms, as hyperæsthesia, paræsthesia, anæsthesia and neuralgia, arthralgia, myalgia especially upon pressure or muscular contraction, dermalgia (Perroud), felt on the posterior aspect of the extremities and trunk, formication and sensation of coldness about the nails (Rendu), etc., etc.

Genito-urinary System.—There are no symptoms pertaining to this system unless there be some complication, as nephritis, diabetes or tuberculosis of kidneys or genitalia. In ordinary uncomplicated cases the urine shows only the changes incident to febrile and wasting affections. It may be scanty and high-colored and on standing may give a deposit of urates and phosphates, but does not contain albumin. Sugar is found in diabetic cases and is then a part of the diabetes, the tuberculosis being secondary.

The *diazo-reaction* of Ehrlich may here be considered. It is obtained as follows: To 40 parts of a solution of sulphanilic acid in distilled water of the strength of 5 c.c. of acid to 1,000 c.c. of water and 50 c.c. of hydrochloric acid, add 1 part of a solution of sodium nitrite 0.5 c.c. in 100 c.c. of water and 40 volumes of urine, after which render alkaline with ammonia

and then shake. If the reaction is present there will be a rose-red
an orange-red ring.

This so-called Ehrlich's test is not diagnostic of tuberculosi
reason that it may occur in enteric fever, certain acute infectious
of childhood and possibly in septicæmia and advanced malignan
The chief value of this reaction in chronic pulmonary tubercu
therefore, in the light thrown by it on prognosis. If persistent
to denote a rapidly progressing form of disease with a not remote te

To this there appear to be exceptions, however (Burghardt).
other hand, as stated by Fraenkel, the absence of the diazo cannot b
as necessarily furnishing a favorable prognosis, since cases of sever
with extensive cavity formation have been seen in which it did
Moreover, as stated by Michaelis, the reaction is independent of
the amount of the sputum and the number of the bacilli.

Tubercle bacilli are not present in the urine of pulmonary tu
unless the case be complicated by the same disease in the geni
tract. Under such circumstances they may be discovered by prope
methods, along with blood and other evidence of renal or genital

Finally it is worthy of note that Crofton has found an abnorm
excretion of calcium in the urine of a tubercularized dog and i
chronic phthisis with cavity formation. He found such an inc
in four out of six suspected cases of latent tuberculosis, which f
quently developed the disease. Crofton attributes this excessive
of calcium to its combination with deutero-albumose which is a pr
produced by tubercle bacilli and found in the urine of tuberculous su

CHAPTER XXVI

PULMONARY TUBERCULOSIS—*Continued*

Physical Signs.—These vary greatly according to the stage of the disease. In the incipiency of the process signs are few and often so indistinct as to necessitate great pains for their detection. In the stage of softening the local evidence of disease becomes plainly apparent, while in a still more advanced stage, when lung destruction is the chief characteristic, the objective marks of the complaint can seldom be overlooked by the most superficial observer.

Inspection. *Early Stage.*—There is but little to be seen in the incipient period, but what there is at this time is highly significant. If the individual uncover his chest completely and stand in a good light it may generally be noted that the two apices are not quite equal both in contour and expansion. On the affected side, the clavicle stands out somewhat more prominently in consequence of slight flattening of the intraclavicular region as well as slight deepening of the supraclavicular fossa. It is noticeable, in addition, that there is slight but unmistakable atrophy of the scapulo-humeral muscles in consequence of which the spinous ridge of the scapula and tip of the shoulder stand forth more conspicuously than on the opposite side. This, together with the flattening of the pectorales in front, produces the wasting of the *shoulder-girdle muscles* which, by many clinicians, is considered so significant a sign of incipient tuberculosis. I have rarely known it to fail. In connection with this atrophic state of the musculature there is deficient expansion of the apex which, although trifling, is yet noticeable on close inspection and forms a very important link in the chain of evidence.

In *confirmed phthisis* the changes just described are more pronounced and may involve even both halves of the chest producing that characteristic form known as the alar or pterygoid chest shown in Fig. 68. The shoulder-blades stand out from the back in consequence of their rotation forward, the arms are turned forward and outward, the chest is sunken and the long, thin neck hangs forward as if not able to support the head. The face has a pale or ashen color, the eyes are bright and the patient presents the appearance designated as the *phthisical habitus.* It requires but a glance to note the emaciation and other signs of a pronounced cachexia. In some cases there may be, in addition, well-marked clubbing of the fingers or the pulmonary osteo-arthropathy described in the chapter on Bronchiectasis.

Palpation.—In the *early stage* this is not likely to afford much information. Pectoral fremitus may not be appreciably altered from that in health

As infiltration progresses, however, the conditions favoring transmission of vocal vibrations become greater and hence the affected apex yields increased fremitus as compared with that of the healthy side. As would be expected, this exaggeration of vocal vibration is especially distinct in the suprascapular region where primary infiltration takes place and, as the disease advances, in the areas of secondary deposits in front. Such increase is apt to be slight, however, and since the right apex may normally, in some individuals, show a more pronounced fremitus, and since the propagation of vocal vibrations depends for its intensity on the loudness and depth of the voice, care and judgment must be exercised in estimating the significance of pectoral fremitus in this early phase of the disease.

In *advanced tuberculosis*, when extensive areas of lung-tissue have become infiltrated, pleuritic adhesions exist and cavities have formed, the results of palpation become correspondingly altered. Over areas of induration without pleural thickening fremitus is increased as a rule, but in some instances it may be actually diminished in consequence of extensive caseation. The presence of dense adhesions decreases fremitus, whereas a vomica may occasion increase of the same, over a circumscribed area. Accordingly, there may be marked differences in the results of palpation in various parts of the same lung. On the whole, I attach but slight importance to palpation as compared with other means of investigation.

Percussion.—In the *early* and especially in the *incipient* stage the results of percussion are likely to be very uncertain. Indeed it is well known that the degree of information obtained by percussion stands in direct ratio to the extent and character of the changes produced by the disease. It is for this reason that many writers attach but small importance to percussion in the commencement of pulmonary tuberculosis. For my part, I believe careful percussion is of the very greatest value at this time, since if very slight impairment of resonance can be detected over a limited zone at one apex it furnishes information of immense significance.

In examining for signs of incipient disease the physician should remember that, as has been previously pointed out, the area of primary tubercle is in the posterior and superior portion of the apex, and hence he is to percuss in the suprascapular region and from this point down the interscapular region rather than in front. With progress of the disease the primary zone of infiltration grows in size and secondary foci appear; so that both the extent and degree of dullness increase, coming to be plainly apparent in front as well as behind. Yet, in all instances, this change in pulmonary resonance is always more pronounced and more extensive over the back.

There is ordinarily no difficulty in the detection of dullness at one apex or possibly at both apices in the cases of confirmed phthisis. It is in the incipiency of the process that the results of percussion are apt to be so uncertain. At this time the examiner must be keen to perceive very slight differences in the pitch of the percussion-note at the two apices and must compare over and over again, if need be, the resonance of corresponding areas. At this stage I would especially urge the careful determination of the height of the two apices. To this end the physician will find it most convenient to stand behind the patient, since in this position he can fit his pleximeter finger most easily and accurately into the depression above the clavicle. Then by means of a dermatographic pencil he can mark the upper limit of each apex. Flint gave the distance to which the tip of the lung extends above the collar bone as from $\frac{1}{2}$ to $2\frac{1}{2}$ inches while Leube gives it as 6 cm., or $2\frac{2}{5}$ inches. If there is incipient tuberculosis of one apex this may generally be demonstrated to be appreciably although but slightly lower than is its fellow. This requires delicate and accurate percussion, yet often yields most valuable information.

Another point to which I attach great importance in the recognition of slight shades of difference in the resonance of the two apices is the following. It not infrequently happens that in the beginning the resonance of the two suprascapular regions seems equal, or so nearly alike, as to place the examiner in doubt. If, now, the patient be required to breathe deeply for a few times it may often be noted that the suspected apex has become appreciably less resonant either generally, as compared with the opposite side, or only in a circumscribed zone. Should this device fail to develop slight inequality in resonance, then the patient may be told to inspire deeply and hold his breath while percussion is performed or let his breath out and then hold it while percussion is again made. This procedure will in some instances aid materially in the detection of an area of impairment which previously seemed equivocal.

Furthermore, there are two periods of the disease in which the resonance may be tympanitic. One is in the stage of excavation when the note over a circumscribed area of greater or less extent, and generally below the clavicle, is tympanitic, cracked-pot or amphoric, as the case may be. There are also the variations in pitch known as the Wintrich Change of Pitch, the Gerhardt Change of Pitch, etc., all of which will be found described at length in the diagnosis of cavity.

The other stage of the tuberculous process in which tympanitic resonance may occasionally be observed is in its incipiency, when one apex is in the condition of alveolar catarrh. Under these circumstances percussion sometimes elicits slight but distinct resonance of a tympanitic quality which, if due care be not exercised, may mislead the physician into thinking the area is not affected. This note precedes the period when resonance becomes distinctly impaired, and hence the physician may be surprised by detecting, a week or two later, unmistakable dullness at the very apex which before was thought free from disease.

Finally, it may be well to point out the fact that in an early stage the changes detected by percussion are limited to one lung and are always more pronounced in the upper than in the lower portion of the chest, unless it may happen that the case is characterized by pleural involvement. In such an event the loss of resonance may be more marked at the base. When in advanced phthisis the second lung has become involved percussion detects all possible shades of dullness in both halves but more extensive and pronounced, as a rule, in the lung that was primarily attacked.

Auscultation.—In the incipient stage the breath-sounds are but slightly altered, yet from the standpoint of diagnosis require most careful and intelligent study. They present all possible shades of difference from slight roughness of quality to faint broncho-vesicular or even bronchial breathing according to the extent of infiltration. Their intensity also may be modified, so that they are exaggerated or diminished as the case may be.

The earlier and more circumscribed the changes in the lung, the slighter and fewer are the auscultatory findings. It may be set down as a general proposition also that the less the impairment of resonance the less do the breath-sounds depart from normal vesicular quality. Accordingly, the stage of incipiency, and still more the stage of latency, is distinguished by such a trifling alteration of the respiratory murmur that it may readily escape attention, especially if the auscultation is restricted to the front of the chest.

In many instances there may be nothing more than roughness of the inspirium with lengthening of expiration. Moreover, these slight changes are usually quite circumscribed to the areas of primary and early secondary involvement, so that they must be carefully sought for in the suprascapular and upper part of the interscapular region when not present in front. Still, the physician will often find that if he auscultates in the three upper intercostal spaces along the parasternal line over the secondary areas of deposit in front, he will perceive a degree of roughness of inspiration that is wanting further outward toward the shoulder. In these various areas the voice-sounds, both whispered and loud, are likely to be intensified.

With progress of disease the breath-sounds become more distinctly roughened and perhaps jerky until, with unmistakable dullness, they become broncho-vesicular or plainly bronchial. Occasionally they are diminished or indeterminate, depending upon the degree of caseation. If râles are heard at this time they are either fine clicks limited to a small zone behind and produced during inspiration or they are an indistinct crumpling. Occasionally there may be a faint sibilus or an ill-defined friction, limited to the apex posteriorly.

An important peculiarity of the fine crepitant râles is their increase in clearness and number during or directly after cough. They are often spoken of as "moisture" and are held to indicate an alveolar catarrh. They are in reality the earliest evidence of beginning change which sooner or later will merge into the stage of softening.

In this *second stage* râles grow more numerous, more distinct, and occupy a larger area. They now are heard as distinct clicks of variable size and are in reality fine bubbling râles. Cough and deepened inspiration bring them out in large numbers and often with almost unpleasant distinctness. They may entirely obscure the breath-sounds, but if these latter are heard they are bronchial. Voice-sounds are bronchophonic or pectoriloquous and in a circumscribed area in the upper lobe they may present a degree of concentration and distinctness very suggestive of a forming vomica. It is not uncommon at this time to get moisture or fine friction-sounds well down toward the base of the lung, while at the apex of the opposite lung the breathing may be harsh enough to suggest formation of tubercles in that situation.

In *advanced phthisis* the auscultatory findings are likely to be made up of a conglomeration of abnormal sounds indicative of the various changes through which the lung has passed in the progress of the disease. At the apex may be bronchial breathing with either copious or scattered clicks. Further down in the scapular region and perhaps extending through to the front there may be an area of variable size over which are cavernous voice- and breath-sounds with gurgling or creaking râles depending upon the activity of the destructive process still going on. While still lower and reaching to the base may be feeble or loud broncho-vesicular breathing interspersed with fine moist crepitations or dry friction-sounds, all significant of the ever-spreading tuberculous and caseating process.

In some cases the tubular or perchance amphoric respiration at the apex and absence of bubbling râles indicate a fibroid transformation, while at the base are the enfeebled breath-sounds and creaking or indefinite rubbing sounds which denote thickening and adhesion of the pleuræ. In the opposite lung chiefly at the upper part may be the obscure bronchial respiration and fine crepitant râles of beginning softening.

In some instances it may be difficult to make out distinct cavities, while in still others the signs of a huge vomica predominate over those of active lung disintegration. Thus the picture is ever varied and yet ever similar, depending upon the stage of the process and its tendency to destruction or arrest. As the march of the disease is from above downward the auscultatory findings are those of an older process at the summit and of a developing and advancing tuberculization and caseation in the middle and at the base of the lung.

Recapitulation.—In the *incipient stage* physical signs are generally confined to one apex and are often very indefinite. They consist of the following: slight muscular atrophy and flattening with possible restriction of inspiratory expansion; slight increase of vocal fremitus in the suprascapular fossa and along the lung-border in front; normal or slightly impaired or tympanitic resonance, the impairment being more pronounced behind; rough and intensified or enfeebled and indeterminate inspiration with prolonged expiration; increase of the loud and whispered voice over a circumscribed area generally in the suprascapular region; no râles.

At a *later period*, but still in an early stage, the signs become more pronounced. In particular, dullness is more apparent but still mainly behind, and râles become audible. These are fine uniformly sized clicks that are heard most clearly during forced inspiration or after cough and are often limited to the extreme apex behind. Areas of secondary infiltration are n likely to be detected by zones of moderate dullness and rough or bronch vesicular breathing.

In the *stage of softening* or moderately advanced stage local signs gro more distinct but are generally confined to one upper lobe or at the mos to two lobes. Muscular wasting and retraction at one apex are pronounced pectoral fremitus is plainly exaggerated yet may be decreased by extensiv caseation; dullness is marked and very likely to extend further downward behind than in front; respiration is distinctly bronchial and loud or enfeebled, while the loud and whispered voice is correspondingly bronchophonic or feeble and distant; râles are present, often numerous and made up of fine and medium-sized clicks that are increased in number and intensity by cough or deepened inspiration.

In the *stage of excavation* physical signs are found in two and possibly three or more lobes and hence the process is no longer unilateral. They consist of the following: general emaciation and cachexia; the alar form of chest; absence of inspiratory expansion of one apex and possibly of both apices or even of an entire lung; exaggerated pectoral fremitus below one clavicle over the seat of a cavity; pronounced dullness over the greater portion of one lung and possibly of both; tympanitic or cracked-pot resonance or Wintrich's or Gerhardt's change of pitch below one clavicle or elsewhere; bronchial, cavernous or amphoric breathing over the upper lobes, as the case may be; râles of various kinds and size over consolidated and caseating areas and over the seat of vomica, gurgling sounds, or the metallic tinkle or only vague crumpling and the cavernous or amphoric voice and whisper; signs of complications as laryngitis, pleurisy, empyema or pneumothorax.

Clinical Varieties of Chronic Pulmonary Tuberculosis.—As I have remarked already, it does not seem necessary to attempt strictly to classify the cases of chronic phthisis ordinarily encountered. It is sufficient to recognize them as chronic. The most of them evince the inevitable tendency to softening and excavation, but this tendency is not equally pronounced in all cases. There is, however, a class of cases in which an attempt at repair by development of fibrous tissue dominates over the inclination to caseation and hence it is well to consider such in a special class.

Fibroid phthisis is the term applied to these cases. The process begins insidiously, as a rule, and the physician is surprised by the discovery of signs of disease involving the greater portion or the whole of one upper lobe. There are marked retraction of the apex, distinct dullness, bronchial breathing and bronchophony, with few if any râles.

In another class of cases the process sets in abruptly, or at all events there is a sudden exacerbation of the local affection. The individual is seized with pain in the upper part of the chest, dry cough and elevation of temperature, and is confined to bed for a week or two. Examination of the chest is pronounced negative or fine crepitating râles are heard at one apex and the condition is called acute bronchitis.

Such, in brief, was the history furnished by a young man but a few days ago. Two years before this quondam college athlete was seized, while swimming, with a sharp pain in the upper front chest. Fever supervened, he took to his bed and summoned medical aid. For two weeks his symptoms persisted, yet his physicians were unable definitely to ascertain their cause, although pleurisy was suspected.

Since that time he has experienced occasional pain in the same situation, especially on sneezing or yawning. Recently an acute illness developed, which was thought to be the *grippe* and for which he was confined in a hospital. Upon convalescing he sought further medical advice and came to me.

He had a temperature of 100.4° F., pulse was 120, and there was frequent but slight cough without expectoration. The right apex was appreciably retracted and there was distinct dullness throughout the upper lobe, most pronounced in the middle of the suprascapular region and in front along the anterior lung border to the fourth interspace. Over this area breath-sounds were semibronchial and accompanied by dry crackling and rubbing sounds that were particularly distinct toward the end of both inspiration and expiration, but not developed or increased by cough. They were therefore considered to be pleuritic friction and not moisture.

Although an opportunity was not given for sputum-examination and the young man refused to enter a hospital for further study of his case with a view to establishing the exact nature of his difficulty, it was yet believed to be an instance of pulmonary tuberculosis which had first declared itself as a pleuritis. It was, I am persuaded, an example of what Fraenkel terms Pleuritogenous tuberculosis that was developing into fibroid tuberculosis of the lung.

I have under occasional observation a young woman who several years ago lost weight and strength, had a dry cough and slight elevation of temperature. At my first examination indefinite dullness and feeble bronchovesicular breathing were discovered at the left apex, chiefly along the inner margin back and front.

Soon thereafter she had a slight hæmoptysis with aggravation of symptoms and was sent to a hospital. From this attack she rallied without difficulty and in time lost her cough. From time to time, however, she complained of sharp stitchlike pain in the left infraclavicular region, when there could be heard vague friction in that area.

At my last examination, now some months ago, the left upper chest was retracted, moderately dull and furnished feeble bronchial breathing and slight bronchophony. In the case of this young lady also there is, I am convinced, a fibroid tuberculosis of the kind described by Fowler and of which I shall again speak.

The case of Mr. E. F. furnishes a typical and striking instance of general fibroid phthisis of the entire left lung. His chest is shown in Fig. 72. This man, a bookkeeper of thirty-one, first took ill ten years prior to my examination.

His illness began with cough, loss of weight, slight pyrexia and other indications of pulmonary tuberculosis. After some months of unavailing treatment in Chicago he went to Colorado where he remained for six years. During the succeeding three years he roamed about from one resort another without added benefit. He then spent one year at home, and the Fall of 1904 sought my opinion regarding another change of climate.

It required but a glance to perceive marked contrast in the contour an diameter of the two halves of the thorax. The inferior portion of th sternum and the adjacent costal cartilages at the left were greatly retracted, producing a somewhat funnel-shaped depression. The entire left chest was flattened and contracted, which nature had attempted to compensate by a left lateral deviation of the spinal column and drooping of the corresponding shoulder. In contrast the right side appeared enlarged. Respiratory movements were abolished on the affected half and the heart was drawn outward and upward.

From apex to base and from median line in front to vertebræ behind the percussion-note was absolutely flat and the sense of resistance was extreme. Excepting beneath the clavicle where breathing was distant and tubular all breath-sounds were absent. No râles were audible and voice-sounds were distant and bronchophonic. Pectoral fremitus was also very indistinct.

In the upper lobe on the right were signs of tuberculous involvement: circumscribed dullness, bronchial respiration and scattered moist clicks. His rather abundant muco-purulent expectoration contained tubercle bacilli and pus-germs.

The man's symptoms exemplified very well those of fibroid phthisis: a somewhat troublesome cough with moderate amount of sputa, a low grade of fever ranging from normal or slightly subnormal in the morning to 100.5° F. or a degree higher in the afternoon depending somewhat on the amount of bodily exercise, considerable emaciation, a rapid, feeble pulse and accelerated shallow respirations which, on exercise, became somewhat dyspnœic.

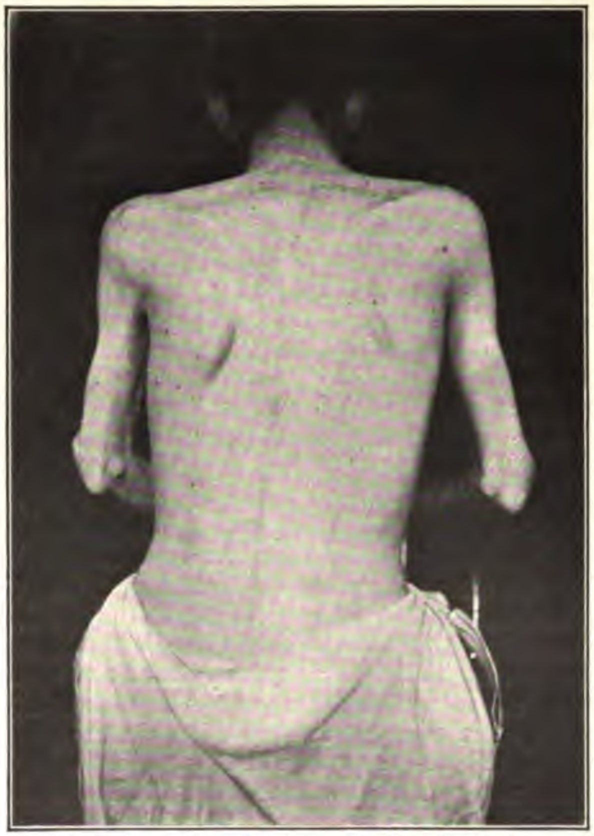


FIG. 72.—Chest of patient with fibroid phthisis.

In this case the process began as an apex tuberculosis which, instead of caseating rapidly, developed into an interstitial thickening of the lobe. In course of time pleurisy with dense adhesions involved and spread through the pleural sac. These fibrinous deposits gradually underwent contraction, the inflammatory process slowly invaded the underlying lung which at length became converted into fibrous tissue.

Whether bronchiectasis had resulted could not be determined, but the character of the cough and expectoration did not indicate their presence. As is usual in such cases the opposite lung had at length become invaded by the tuberculous process, and it was this rather than the fibrous lung which threatened to hasten the downward progress of the case. It was subsequently learned that the man went to San Antonio, Texas, and there became acutely ill with high fever and corresponding intensification of all his symptoms.

This last case corresponds to Sir Andrew Clark's tuberculo-fibroid phthisis, since the process began as a chronic pulmonary tuberculosis and, owing to unusual resistance on the part of the tissues, passed into a condition of fibrosis instead of extensive caseation. It is sometimes puzzling to decide whether such cases are or are not really tuberculous, particularly when the opposite lung does not evince signs of tuberculosis and the purulent, possibly fœtid, sputa do not contain bacilli. These are the cases which Moxon designates as "phthisis of which the history has been forgotten."

Fowler, in remarking on the fibroid variety of pulmonary tuberculosis, states that there may be such an absence of symptoms and physical signs as to render the physician doubtful whether the case is really one of tuberculosis at all. He says further that in these cases the "distinctive pathological feature is the fibroid transformation of miliary tubercles which do not reach the stage of either caseation or softening." Fowler would distinguish these from the cases described by Clark and illustrated by the case of E. F. given above.

The instance of the young woman whose case has been briefly outlined on page 500 has often been puzzling on account of the paucity of symptoms and pulmonary findings, and hence has seemed to me to answer to the class to which Fowler applies the term fibroid. As time has gone on the signs have grown rather more pronounced, but for the first two or three years I was often at a loss to recognize any definite local signs, while her symptoms never consisted of anything more than persistent thinness, occasional transient elevation of temperature to 99.5° or 100° F., and a trifling cough without sputum or only a scanty amount of mucus.

Bacilli have never been discovered, although, as must be confessed, they have not been often sought for. She has repeatedly experienced pleuritic pain at which times vague friction has been detected, and with the lapse of years definite evidence of retraction has appeared. I confidently expect an acute outbreak of tuberculosis in time but thus far natural resistance has triumphed, in part at least.

The *symptoms of fibroid tuberculosis* of the lungs are slight so long as the disease remains limited to the one lung or complications do not supervene. In some instances they may, at the beginning, be such as mark any ordinary case of chronic ulcerative tuberculosis, but after a time cough and expectoration grow less, fever lessens, strength improves and the apex disease takes on a less active character. The cavity, if one have formed, becomes surrounded by dense interstitial tissue; or a caseous focus becomes encapsulated and tuberculous nodules undergo fibrous or calcareous transformation. The upper lobe in which such lesions are seated shrinks, yields a dull note and bronchial respiration with only superficial creaking and bronchophony or even cavernous voice over a circumscribed area.

In this state of things the process remains for many years and the individual considers himself practically well. I have such an instance in mind, as I write, in a prominent business man of Chicago. After a longer or shorter period, however, there comes an exacerbation of symptoms and thenceforth signs of moisture appear below the fibrous area, or tubercles form in the opposite apex, and the patient eventually succumbs to a more or less rapidly progressive tuberculosis of ordinary features. In other cases the prolonged expectoration of pus is finally followed by amyloid disease of liver or kidneys, or an acute miliary tuberculosis terminates the career.

In this connection must be mentioned certain cases of fibroid tuberculosis which develop on top of pulmonary emphysema and may even appear to be only instances of the latter disease. Fraenkel refers to such and gives credit to Sokolowski for having especially called attention to them under the name of *Phthisis fibrosa ab emphysemate.* Instances of the kind are frequently admitted to the Cook County Consumptive Hospital at Dunning and it is there that my attention has been called to them by the medical superintendent. As seen at this institution these patients are generally old men with typical barrel-shaped chests who furnish no history suggestive of tuberculosis, but rather of emphysema and chronic bronchial catarrh.

On admission they are found to have slight elevation of temperature to 99.5°, 100° or 100.5° F., a troublesome cough with muco-purulent or purulent expectoration that may now and again be blood-tinged. The physical signs are those of emphysema with rhonchi but no definite dullness. The sibilant râles are, however, apt to be more marked in one of the lungs or at one apex. The heart is dilated, often with a systolic murmur, the arteries are stiff and perhaps tortuous, the veins prominent and the pulse accelerated and weak.

The patients suffer from dyspnœa and emaciate rapidly which, by reason of their large bony framework and large round chests and sunken bellies, causes them to present a very striking picture (see Fig. 2). The degree of emaciation and the other symptoms become highly suggestive of tuberculosis, but the sputa may be and often are examined scores of times before bacilli are discovered. Likewise repeated and painstaking examinations of the lungs are made before apical signs of disease are discovered.

When at length such findings are determined they consist of vague dullness with marked resistance at one or possibly both apices behind, in the suprascapular region and running downward along the inner border of the scapula. Over this impaired and resisting area breath-sounds are feeble, seem semibronchial or harsh rather than tubular, and are likely to be accompanied now and then by faint whistling râles or a few crepitations after cough. Vocal fremitus may be somewhat increased and the voice is moderately bronchophonic. Occasionally the signs and character of the sputa suggest the presence of bronchiectases.

The course is usually very chronic and the patients keep up and around, declaring they would be pretty well were it not for their cough and breathlessness, which keep them awake at night. The development of œdema or not depends upon the degree of circulatory embarrassment.

Tuberculosis in Childhood.—Inasmuch as this work deals with disease of the lungs and not of the general system, I shall consider in detail only the pulmonary manifestations of tuberculosis in children, and refer but briefly to such other points as are thought necessary to a full understanding of the subject.

The disease is probably more frequent in infants and young children than is recognized and largely because it masquerades in forms which make it simulate other affections. If it affects the lungs at all in the earliest years of life it does so only as a part of a general infection or secondarily to tuberculosis of the bronchial glands.

In infants, that is, up to the age of two and a half years, it is the lymphatic system which bears the brunt of the disease, and hence it is that in babies tuberculosis is very apt to occasion symptoms embraced under the terms marasmus, tabes mesenterica and tabes scrofulosa. The explanation thereof is probably to be found in the manner of infection at this early age, the bacilli being taken into the system either with cow's milk, as maintained by von Behring, Fürst *et al.*, or along with contaminated articles introduced into the mouth.

Infection by inhalation may also take place but if so the bacilli pass into the lymphatic system and reach the bronchial glands whence, in time, they invade the lungs secondarily (Cornet *et al.*). Other possible channels of infection have been already considered (see Modes of Infection) and need not here be mentioned. However the germs may reach the lymph nodes they are, owing to the great activity of lymphatic circulation in children, readily transported to various parts of the body.

Accordingly it is common for children to manifest bone tuberculosis and tuberculosis of the meninges, the former not generally making itself known until after a number of years, and then often independently of demonstrable involvement of other organs or structures. Meningitis, on the other hand, is very likely to appear as a part of a general tuberculosis although it not seldom so dominates the scene as to be the only form of the disease recognized.

Another mode by which tuberculosis becomes general in children as well as in adults is by the rupture of a caseous gland into a blood-vessel and the consequent dissemination of the bacilli throughout the entire circulatory system. Yet, whatever be the precise manner of infection in infants, the disease most commonly manifests itself as a chronic progressive wasting, either without fever or with a remitting type of pyrexia.

The little ones present an expression of intense weariness mingled with patient resignation that is truly pathetic and cannot fail to attract attention. The course of the disease as thus briefly sketched is usually slow and follows [illegible] upon some acute infection as measles, pertussis or influenza. The [illegible] and debility often set in without any recognizable cause [illegible] to have no apparent date of commencement.

[illegible] cases the tuberculosis manifests itself as a general [illegible] may be either acute or subacute, according to the rapidity of its course. This form results from the discharge of a multitude of bacilli into the general circulation by which they are borne to all parts of the body.

The *acute miliary tuberculosis of children* presents a striking contrast to the chronic generalized form since its course is usually stormy and short. General symptoms may predominate over the pulmonary and then are characterized by high continuous fever or by a temperature-curve showing pronounced irregularities, by prostration, abdominal distention, splenic tumor, and a tendency to delirium which give the disease a typhoid-like character.

In other cases there are râles and indefinite areas of dullness which cause the disease to be mistaken for broncho-pneumonia. Cyanosis becomes marked, yet notwithstanding the very manifest general bronchitis there is but slight cough. There may be periods of apyrexia which delude the friends into the belief that the disease is abating.

The *subacute form* of miliary tuberculosis of childhood is distinguished from the acute by its more protracted course and by the fact that periods of high fever may alternate with times of apyrexia. Accordingly it is assumed that the infection takes place intermittently through the occasional discharge of bacilli into the blood-stream. Fraenkel is authority for the statement that, according to Henoch, a suspicion that this form of miliary tuberculosis is developing may be entertained if there occur short periods of high and unaccountable fever, possibly of only a few days' duration, followed by other periods, even by weeks, in which the temperature is perfectly normal, and if examination of the lungs reveals areas of exaggerated breathing with possibly a few catarrhal râles.

Under the term Typho-tuberculosis is described by Landouzy, Jeannel, Papon and Marfan a form which corresponds with the subacute variety just portrayed and which may be referred to the discharge periodically, as it were, of bacilli into the circulation, but in moderate numbers so that the general system is able to resist successfully, and recovery eventually occurs.

The clinical picture is very like that of enteric fever, being characterized by pyrexia showing daily exacerbations and remissions for eight to ten days, after which it declines by degrees. About this time the lungs display moderate dullness, chiefly toward the summit rather than the base, with diminished or semibronchial breathing and fine bubbling râles. The disease differs from typhoid fever in the absence of rose spots and in the less serious involvement of the general system. In the course of time even the pulmonary symptoms may disappear and recovery is complete.

Pulmonary tuberculosis in young children exhibits some peculiarities that distinguish it from the disease at a later age. In the first place, it usually forms but a part of generalized tuberculosis during the time of the first dentition and often until the fifth year of life. In the second place, it is associated with and probably is secondary to disease of the bronchial lymph nodes. On this account its distribution in the lungs is different from that in adults.

The primary location is not at the apex, as is the case in adults, and as has been pointed out repeatedly in this work, but the growth of tubercle takes place in the middle and lower portions of the organ, so that signs of the disease are discoverable behind, about the hilus of the lung and in the lower lobe. Such is the case, at all events, before the fifth and probably before the seventh year of age. From this time onward the affection tends more and more to assume the characters seen in persons of mature growth.

From the bronchial nodes the disease may invade the lungs in either of two ways: (1) The indurated, agglutinated and often caseous glands at the root of the lungs may become united to the pleura by inflammation and through the lymph channels disseminate bacilli in that portion of the parenchyma contiguous to the hilus of the lung, that is, in the middle of the lung. (2) A caseous and softened gland may discharge its contents into a bronchus and thus set up a tuberculous broncho-pneumonia in the lower lobe which process may be confluent or discrete.

On the assumption that pulmonary phthisis in young children is secondary to bronchial gland tuberculosis the peculiar localization of the process is readily intelligible. Yet it is interesting to note the explanation given by Birch-Hirschfeld of the relative immunity of the apices in the early years of life as contrasted with the predilection of these in adults. His reasons, as stated by Fraenkel, are based on anatomical and physiological grounds.

As has been pointed out in considering the primary seat of apical tuberculosis, Birch-Hirschfeld attributes the vulnerability of the upper lobe to the arrangement of the bronchus leading to this lobe which hinders perfect ventilation of the apex and favors the deposit of bacilli on the wall of the apical air-tube at that point where the feebler current of air in its outward passage is encountered and arrested by the stronger stream passing along the tube into which the apical bronchus merges. In the early years of life this peculiar anatomical arrangement of the air-tubes does not exist. Furthermore, the chest of the child is far more elastic and mobile than is that of the adult, and hence the apices are more vigorously inflated with each respiration and better ventilated.

Prior to the fifth and perhaps to the seventh year pulmonary tuberculosis is peculiar not alone in its location but also in its clinical manifestations. With advancing age these differences tend to disappear until, when a child reaches the age of ten or thereabouts, the disease assumes the characters seen in the adult. From a clinical standpoint the first thing to be borne in mind is that the lungs are likely to show disease only as a part of a generalized process. The second is that in young children pulmonary tuberculosis is rarely chronic but usually runs an acute course.

In the first five years of life the tuberculosis of the lungs generally develops in the wake of measles, whooping-cough or influenza, and taking the form of a bronchitis with scattered areas of dullness, bronchial breathi and subcrepitant râles, is regarded as a secondary broncho-pneumonia eith acute or subacute. If a suspicion of its real nature be entertained, t diagnosis is difficult or impossible on account largely of the fact that sput are swallowed and hence cannot be obtained for microscopic examination The little patient displays a more or less rapid and resistless increase o weakness and pallor, notwithstanding a fairly good appetite and considerable amounts of food. Cyanosis and dyspnœa develop and may be out of proportion to recognizable signs of disease within the chest.

Cough varies in frequency and severity but is generally a constant symptom. *Hæmoptysis* is a rare occurrence in young children and yet has been noted. Henoch is said to have seen it only about a dozen times and in two instances which proved fatal found it due to rupture of a small pulmonary aneurysm the same as in the adult. If the sputum is needed for diagnostic purposes the child may be inverted when in the act of coughing or, as suggested by Fraenkel, a probe wrapped with sterile cotton may be introduced into the pharynx during a coughing fit in the attempt to catch the expectoration as it leaves the larynx.

Diarrhœa may be the expression of an intestinal catarrh and may result from the swallowing of the germ-laden sputa, or may be due to intestinal tuberculosis. There may be enlarged cervical or other lymph nodes, particularly within the abdomen, or there may be disease of some joint or bone, which conditions will aid in establishing a correct diagnosis. There may also be a history of tuberculosis in some other member of the family.

The *chronic form* of pulmonary tuberculosis in children bears a more or less close resemblance to that in persons of maturer growth and was well shown by the case of a seven-year-old girl first brought to me from the interior of Illinois in December, 1903.

The child had had a severe cold the January previous and since that time had not appeared well, especially during the late Fall. The symptom that caused the mother most uneasiness was a cough and at night this was especially troublesome. There was no visible expectoration as the sputa were swallowed. The child appeared languid, did not eat well, looked paler than normal, but was thought not to have lost weight. There was a little fever at times but this had not been a marked feature. One sister and one aunt had died of pulmonary tuberculosis.

Physical examination corroborated the mother's statements regarding the state of the nutrition and temperature, the latter being 99.2° F. in the mouth. The girl was plump and looked like anything but a consumptive. Partly for that reason and because of the uncertain character of the pulmonary findings the case was at first thought to be one of chronic bronchitis with areas of collapse.

At the right apex in front resonance was impaired and breath-sounds were feeble and accompanied by an inspiratory sibilus. From the fourth to the sixth rib and from slightly within the nipple to the anterior axillary line dullness was more apparent with bronchial breathing and fine râles on deep inspiration. Behind there was quite marked dullness in the right interscapular region and breathing was bronchial. At the left front from second to third interspace resonance was impaired and breath-sounds were harsh. At the left base outside the scapula and from sixth to ninth rib there were rhonchal fremitus, slight dullness and catarrhal râles.

In April, 1904, the child was brought to me again, not having improved, when it was easy to determine that the areas previously found had grown more dull while the breath-sounds were plainly bronchial. Tuberculosis was now diagnosed and upon getting the girl to expectorate a little sputum and subjecting this to microscopic examination two or three unmistakable bacilli were discovered.

In this case the dullness in the right interscapular region indicated the changes immediately adjacent to the bronchial nodes at the root of the lungs, while at the base on both sides were secondary localizations. The apex itself was comparatively resonant and free from the auscultatory findings so commonly found in persons of an older age.

In the following case, a girl of thirteen, the disease was manifested in its ordinary manner: B. H., a Jewish schoolgirl of thirteen, was brought to me March 21, 1904, by her physician, Dr. Josephson, because of hæmoptysis and other symptoms of pulmonary disease. Her first hæmorrhage had occurred nine days previously and had persisted for two days. On the morning of the 21st she had again coughed up considerable blood. As regards family history it was stated that her mother had died of pulmonary tuberculosis, as had a maternal uncle. Herein lay the source of the child's exposure to infection.

Emaciation was not marked and the temperature was not high, 99.4° F. in the mouth. The pulse was 112 and weak. Upon examination the apices were moderately retracted and on percussion and auscultation showed the localization found in adults. The right upper lobe was dull, both front and back being especially intense below the clavicle, with bronchial breathing and occasional medium-sized clicks. At the right back below the eighth rib were also dullness, bronchial breathing and fine bubbling râles, indicating an area of secondary broncho-pneumonia very likely from aspiration. In the opposite side, both front and back, were findings of a less pronounced character, yet in the suprascapular region moist râles were numerous.

I could not determine the source of the blood, but it was plain that the disease had passed from the first into the second stage as shown by the extent of lung involved, if by nothing else. The cervical and axillary glands were indurated and it seemed likely that the pulmonary affection was but a part of the disease, the lymphatic glands having become previously infected through contact with her mother.

Tuberculosis of the glands in children is so common that, as Henoch has said, one can reckon with certainty that wherever there occurs in the body a tuberculous or caseous process, there will the lymph nodes be found correspondingly affected. Thus, according to Fraenkel, of 302 autopsies on tuberculous children Steiner and Neureutter found the lymph glands diseased in 268. Accordingly, it is fair to assume that when the lungs are involved in children there is also tuberculosis of the bronchial glands. Occasionally these structures alone are involved and produce symptoms referable thereto. Yet, of Steiner's and Neureutter's cases in only 36 were the tracheobronchial nodes alone affected.

The symptoms occasioned by tuberculosis of these glands depend upon the degree of enlargement they have undergone and consist essentially in pressure-effects on veins, nerves, trachea and air-tubes or gullet, with dullness in the bronchial-gland area around the hilus of the lungs. If a softened gland ruptures into a bronchus, areas of aspiration pneumonia result, with symptoms of pulmonary tuberculosis and corresponding physical findings.

Tuberculosis of the Lungs in the Aged.—It is commonly believed by the laity and by the profession that old persons seldom fall victims to consumption. In a certain sense such is true and yet it would be very erroneous to suppose that old age affords any immunity to the disease. As a matter of fact, Cornet's statistics show conclusively that pulmonary tuberculosis is relatively very frequent after the fiftieth year, being conspicuously common between sixty and seventy.

Thus, it is shown that 1 out of every 43 males and 1 out of every 62 females living at this age is tuberculous, whereas between fifteen and twenty years of age, the period of life at which consumption is universally recognized to be great, only 1 out of every 200 living males and 1 out of every 175 living females is tuberculous. Other tables compiled by Cornet bear out these figures.

It must not be supposed, therefore, that the absolute number of consumptives among the aged is greater than during the early adult period, for such is not the case. The total number of tuberculous aged is small, but because the total number of persons who have reached old age is small, the percentage of such who are tuberculous is relatively high. In other words, this all goes to prove that human beings may succumb to tuberculosis at any age provided conditions of infection are right.

The localization may be primary in the lungs of the old as well as in young adults, or it may be secondary to tuberculous lesions in other parts as the bronchial glands. The symptoms are not essentially different from what is seen in ordinary cases of phthisis, except that the process is said by some to display a rather greater tendency to chronicity. In some cases the special features of the disease appear to be those of bronchitis and for that reason are likely to mislead.

In other instances the pulmonary signs are disguised by the emphysema and, unless the sputa are examined microscopically, may be held to indicate nothing more than emphysema with chronic bronchitis. In still others caseation and excavation proceed apace and are accompanied by emaciation and marked debility the same as in any other case at a different age.

Aged individuals do not react vigorously, as a rule, to any disease, e. g., pneumonia, but exhibit a striking tendency to prostration. Accordingly, in the disease now under consideration one would scarcely expect as high body-temperatures as are seen in the young. They show a peculiar tendency to catarrhal disorders and this, together with the senile atrophy of the lungs so often found in the aged, probably accounts largely for the fact that pulmonary tuberculosis in the old often assumes the characters of a chronic bronchial catarrh.

In two cases I saw some years ago, one a man of about seventy, the other a man of sixty or thereabouts, as I now recall, the disease showed itself as a fairly uniform broncho-pneumonia of the upper lobe. There were the ordinary signs of dullness and copious fine and medium-sized râles which almost entirely obscured the breath-sounds. In both, the fairly abundant muco-purulent expectoration contained bacilli, fever was moderate but lack of strength was a marked feature, especially in the one of seventy in whom the disease was the more advanced. In short the disease displayed no characters especially different from those of any ordinary case of consumption.

Complications.—It is possible, of course, for a case of pulmonary tuberculosis to be complicated by intercurrent affections having no direct relation to the primary disease. It occasionally happens also that some symptom, e. g., hæmoptysis or a peripheral neuritis, becomes so troublesome or alarming that it may in a sense be looked upon as a complication. Such, however, are not what are ordinarily embraced by the term complications of consumption. These are for the most part manifestations of the tubercular process in other parts than the lungs and with one or two exceptions will be so considered here.

Laryngeal Tuberculosis.—Among the more common and serious complications of pulmonary tuberculosis is tuberculous laryngitis. This may now and then be encountered so early in the course of the pulmonary complaint or may so outweigh, as regards the patient's comfort, the manifestations of lung mischief that it may even seem to be the primary trouble and may obscure the lung disease for a time. In the great majority of instances, however, the larynx becomes involved comparatively late in the course of the pulmonary affection. At other times the laryngitis comes on relatively early but sufficiently long after the incipiency of the main trouble to make it very evident that it is secondary.

It is unnecessary to enter into an elaborate description of the changes constituting laryngeal tuberculosis, since they can be found in works devoted to diseases of the upper air-passages so much better than I could give them here. It will suffice to state that at first the change consists in an infiltration either of the interarytenoid space or of one of the false or true cords in consequence of which the voice is thick and husky. The inflammatory swelling also excites or aggravates the cough but does not interfere with deglutition.

Later on in the process ulceration is likely to result, when the voice may become whispering and cough may be almost incessant and result in the expulsion of a small amount of tenacious mucus and pus that is apt to contain bacilli. When the epiglottis becomes ulcerated, as is likely eventually to occur, deglutition is painful and the condition of the consumptive is truly pitiable.

It must not be supposed that hoarseness of voice is necessarily an indication of tuberculous implication of the larynx, for it is often due to simple laryngeal catarrh. The more serious involvement of the larynx is generally attributed to infection in consequence of the passage over its mucosa of bacilli contained in the expectoration, but inasmuch as not all persons whose sputum carries bacilli with it contract tuberculosis of the organs of phonation, it is presumable that in some cases the bacilli are more virulent or the laryngeal mucous membrane is more vulnerable. This complication is especially unfortunate as it is not very amenable to treatment and hence hastens the downward progress of the pulmonary disease.

Pleuritis.—Another and even more frequent, but generally less serious, complication of chronic pulmonary tuberculosis is pleurisy. It is very common for the consumptive to complain of pain in one side of the chest which interferes with inspiration and renders cough ineffectual. At the same time he feels ill and usually displays an elevation of body-temperature together with increased frequency of the pulse. If the chest be examined a friction-rub is likely to be detected on the side corresponding to the pain but not necessarily to the seat of pulmonary disease. The symptoms are the result of a dry, circumscribed pleurisy.

Such an attack is usually shortlived and in a few days or a week the individual feels about as well as before. Such pleurisies are not to be lightly regarded, however, for they may indicate a spread of the tuberculous process and if often repeated may, in time, materially hamper respiratory movements. Moreover, in the case of a very feeble patient they may prove the straw that breaks the camel's back. It is also important for the practitioner to remember that sudden sharp chest pains may be due to intercostal neuralgia and not to pleurisy, and hence possess a different prognostic significance. They are to be differentiated by the absence of pyrexia or increase of the fever already existing and by the want of friction-sounds.

Another form of pleuritis sometimes seen in the course of chronic phthisis is that with effusion. In my experience sero-fibrinous pleurisy has not been so frequent late as early in the disease, but that such may occur is amply attested by the experience of others. The exudate may be serous and of the usual appearance and specific gravity (1.15 or higher) of such an inflammatory effusion, is rich in lymphocytes or it may be blood-tinged and, according to Cornet, it is rarely very abundant. In the cases I have seen there have seldom been signs of extensive or far-advanced caseation. The evidences of apex disease have usually been limited and not always confined to the side showing the effusion. Indeed they have often been so slight, especially when in the lung compressed by the fluid, as to render difficult a positive conclusion concerning the state of the apex.

Such effusions rarely necessitate aspiration and in fact it is an open question whether they do not serve to hinder the progress of the lung-mischief by restricting respiratory movement in the same manner as does artificial compression of the lung. Rapid absorption of such a serous effusion may be followed by acute miliary tuberculosis (Litten) or by tuberculous disease of bones or joints (Czerny). Cornet is of the opinion that the pleurisies complicating pulmonary tuberculosis may result from the action of toxins carried to the pleura without the formation of tubercles.

Empyema.—Another variety of pleuritis that is generally regarded as a most serious complication of chronic pulmonary tuberculosis is that attended with suppuration or, as it is called, empyema. Such an exudate is usually due to infection of the sac from a superficially located caseous focus. It may be purely tuberculous when, as regards symptoms, it is of the nature of a cold abscess, or it may be septic and caused by invasion of the pleural cavity by pus-germs.

A purely tuberculous empyema may arise insidiously and produce no symptoms aside from those of pressure, and as the exudate is not often large and may be circumscribed the signs of pressure are seldom severe. Consequently a consumptive with this form of empyema may even be in fairly good nutrition and apparently none the worse for his carrying about a pint or more of innocuous pus in his pleural cavity.

Now and then persons are seen who present a fistulous opening in the chest-wall through which pus is constantly discharged and such a perforation may be at a considerable distance from the actual seat of the empyema. Apart from the inconvenience of such a discharging fistula these individuals do not seem to be any the worse for this local lesion. They do, however, run the risk of amyloid disease of some of their other viscera in the course of time.

It is an entirely different affair when an empyema is septic. Then there are chills, fever, sweats and profound prostration to which the individual will inevitably succumb if not relieved by the surgeon. More than one patient whose condition was desperate on this account has been saved by timely interference and ultimately restored to comparative health.

The contrast presented by these two forms of empyema makes very pertinent the question, Should every case of empyema occurring in pulmonary tuberculosis be subjected to operation by rib-resection? As a result of an extensive experience with such cases S. G. Bonney has expressed the opinion that cases of what may be termed innocuous tuberculous empyema do not necessitate such a radical operation. This is on the theory that it "is necessarily followed either by a sudden expansion of the previously compressed lung, which affords opportunity for renewed activity of the tubercular process, and usually results in very quick softening and cavity formation, or is attended by failure of the lung to expand and obliterate the pleural cavity, which means long-continued pus formation and great danger of amyloid change. In the absence of such clinical indications as fever, sweats and chills, it seems exceedingly ill-considered, not to say foolhardy, to precipitate the patient into the midst of such peril." With the foregoing I am in full accord from theoretical considerations.

Pneumothorax.—A very serious and, in greatly reduced consumptives, an often fatal complication is escape of air into the pleural cavity, i. e., pneumothorax. It results usually from the rapid softening of a caseous focus in the periphery of the lung and consequent perforation of the visceral pleura, or from rupture of the thin, perhaps caseous, layer of lung overlying a cavity situated close beneath the sac.

The most common location of such a communication between the pleural cavity and a vomica or air-tube, is on the anterior surface of the lung, between the second and fourth interspaces and the mammillary and anterior axillary lines. The symptoms usually develop rapidly after a forced inspiration, a violent fit of coughing or the lifting of some heavy object, and consist of shock and dyspnœa. They may, in exceptional instances, come on gradually. The most urgent dyspnœa which, if not relieved by aspiration, may amount to actual suffocation is witnessed in the variety known as *valvular*, in which a flap of membrane permits the ingress but prevents the egress of air, so that the sac rapidly becomes overdistended and the lung and adjacent viscera are correspondingly compressed. When, on the other hand, the rupture remains open there may be no very distressing symptoms.

Other phenomena than those of mere mechanical pressure depend upon whether air alone enters the pleural cavity or the sac becomes invaded by bacillary toxins or pus-producing organisms. In the majority of cases serum or pus is found in the sac along with air and we have hydropneumothorax or pyopneumothorax. For the physical signs of these conditions the reader is referred to the appropriate chapter.

The prognosis of this complication depends upon the rapidity of its occurrence and upon whether air alone or air and pus are contained in the pleural cavity. A very feeble consumptive in the last stages of his disease is likely to die soon as a result of the complication. When, on the contrary, the pneumothorax develops slowly or the air is not contaminated by germs there is a possibility of the patient's surviving the mishap and even of his not being much the worse therefor. The latter is the less likely proposition, however.

Intestinal Tuberculosis.—This complication has been considered already in analyzing the symptoms of chronic pulmonary tuberculosis and hence but little need be said here. It manifests itself usually as diarrhœa with cramp-like pain and tenderness. The laity and physicians alike are wont to regard any obstinate diarrhœa occurring in the course of consumption as tuberculous. Caution should be exercised, however, in so regarding looseness of the bowels in this complaint, since fermentative indigestion is common among tuberculous subjects and such fermentation may readily set up diarrhœa. This form is amenable to dietary regulation and herein lies a therapeutic differentiation.

On the other hand the diarrhœa produced by and symptomatic of intestinal ulceration is a most intractable one and can rarely be arrested for more than a very brief period by restriction in diet or medicines. The dejections are thin and of a brownish color and prove very exhausting. Indeed, it is in this respect mainly that this complication is so serious and forms such a cause for dread to the practitioner. Coming on as it generally does late in the course of the primary malady it contributes greatly not only to the discomfort but also to the death of the consumptive.

Intestinal tuberculosis is seen most commonly in individuals who have been in the habit of swallowing their expectoration and hence consumptives should always be warned against such practice.

Anal Fistula.—This is another complication of some cases which is very apt to be looked upon as tuberculous. It begins in infection and subsequent ulceration of the lymph node situated just inside the anus. This leads in time to a sinus and when this has burrowed its way through to the exterior produces a fistula. Experience teaches that such a fistula occurs in persons who are not by any means tuberculous and hence it is not at all inevitable for it to have such an origin in consumptives. Neither does its presence signify that it is a part of a general intestinal tuberculosis. As a matter of fact, anal fistula is not common among consumptives. Fox is authority for the statement that Cotton found it but 3 times out of 1,000 cases, Hayden but 3 times in 330 cases, and Spillmann but 523 times out of 14,730 cases of phthisis. Pollock met with it 31 times, and of these 27 occurred in males, which surely must have been fortuitous as it seems to me.

Fistula in ano has been thought to affect favorably the course of consumption by serving as a derivative from the lungs. Hence both patients and doctors are sometimes loath to remove it by surgical methods. Such a belief is not well founded, however, any more than the notion that a fistula does not heal well. If treated before the person is too reduced it will generally heal, but operation is not justifiable when the consumptive is too prostrated to bear the shock of operation or the depression of an anæsthetic. The local use of cocaine would scarcely be warranted in a feeble consumptive, and moreover if he were far gone in his disease there could be nothing gained by subjecting him to operative interference.

Tuberculosis of bones, genitalia, joints, meninges, peritoneum, etc., may also prove serious, even fatal, complications. At this present writing there is an inmate of the Cook County Consumptive Hospital, a man of twenty-nine whose lungs present dullness, bronchial breathing and râles in both upper lobes, while scattered throughout the remainder of the chest are numerous subcrepitant and creaking râles which, with high temperature and hyperresonance at the bases, suggest acute miliary tuberculosis.

In addition there is a spondylitis of the last two dorsal and first lumbar vertebræ, while a psoas abscess, that probably owes its origin to the spinal tuberculosis, occupies the left iliac fossa and extends beneath the integument of the back over the left half of the sacrum.

I recall a young man with advanced pulmonary phthisis who had a chronic otitis media that occasioned considerable annoyance to himself and friends by the abundance and fœtor of the discharge. It had existed prior to the outbreak of his pulmonary symptoms and probably owed its origin to an entirely different form of infection.

Likewise one may encounter complications arising from causes wholly independent of the primary disease. Occasionally, on the other hand, one may have to deal with complications concerning whose connection with the tuberculosis much doubt may exist. Thus, I have recently seen a consumptive who developed acute arthritis of the left ankle, knee and shoulder. Seeing him but the once after the onset of the joint inflammation I was in doubt whether this was an intercurrent rheumatism of accidental origin or a streptococcus arthritis depending on his mixed infection and hence a manifestation of a bacteræmia. Cultures from the blood would have been interesting and possibly instructive, but for certain reasons could not be made.

Whatever its nature, such a complication in an advanced case of phthisis is certainly serious. In the case mentioned it occasioned pronounced rise of temperature to nearly 103° F., and a consumptive already reduced by his primary disease cannot withstand such an added strain on his vitality.

Syphilis is another disease which it may seem strange to reckon among possible complications. Experience teaches, however, that this complication is not so infrequent as might at first sight be expected. This has been impressed upon me recently by the case of a young man whom I sent away a year ago on account of an active pulmonary tuberculosis. After this patient had recovered from an intercurrent sero-fibrinous pleuritis, he contracted a chancre.

This gave him a sore throat which yielded only to specific medication and, until its real nature was ascertained, proved in itself a serious complication. When I saw him he had a skin eruption which was undoubtedly of specific origin although it did not appear until his physician, C. L. Minor, had instituted tentative treatments with Maragliano's antitubercle serum. The young man was put upon vigorous specific treatment, but as he was seen by me only three weeks ago it is too soon to predict what will be the effect of this added infection upon his tuberculosis. It might be naturally supposed that it would aggravate the primary disease. There are reports of cases showing the association of the two infections which appear to bear out the belief of a natural antagonism between the two.

In an interesting and suggestive paper on the "Association of Tuberculosis and Syphilis," read by Dr. F. I. Knight at the Niagara meeting of the American Climatological Association, reference was made to reported instances of this combined infection which go to prove that syphilis may exert a curative influence on a preëxisting pulmonary tuberculosis. R. Abraham, F. W. Ross, Pourtalis and M. I. Monteverdi have all recorded such cases.

Pourtalis reported twelve cases of second-stage phthisis which contracted syphilis, and in all of them the tuberculous process was arrested and appeared to be completely cured. On the basis of these instances Pourtalis formulated the following conclusions:

(1) Syphilis engrafted on a phthisical patient arrests the progress of the phthisis. (2) Syphilis engrafted on a phthisical patient becomes of a mild type. (3) The antagonism of the bacteria of syphilis and phthisis should cause their toxins to neutralize each other. (4) When the bacterium of syphilis is discovered and cultivated, and its toxins obtained, a serum may be obtained by inoculation with which consumptives may be rendered immune and cured. (5) We believe that the inoculation of the blood-serum of patients in the third stage of syphilis may be of much benefit to the phthisical.

In opposition to these sanguine views of Pourtalis, Bernheim concluded from a study of clinical reports of 43 cases of such an association that the effect of the specific infection varied according to the stage of the tuberculosis in which the chancre was contracted. The influence of the syphilis is almost unnoticeable in the prebacilliary and first stage, whereas the effect is deplorable in consumptives greatly weakened by the ravages of the phthisis. Thus Stieffel narrated the case of a woman who contracted chancre after having had pulmonary tuberculosis for four years. This latter became worse at once; she could not tolerate antisyphilitic treatment and soon succumbed to the phthisis.

It is also interesting to note the effect of tuberculosis upon a preëxisting syphilis. According to Jacquinet, Stieffel, Pidoux, Galliard, Fabry and others the first and second stage of syphilis are greatly aggravated by the advent of tuberculosis, whereas in the tertiary period the course of both is mild, owing probably to the tendency to fibrosis in this stage (Knight).

The reason for the unfavorable influence of tuberculosis upon syphilis in the first and second stages of the latter is thought due by Fabry to the intolerance for specific medication under such conditions. Not only must the mercurial salt be injected hypodermically in small doses and at long intervals, but the patient must receive cod-liver oil and the best of hygienic management.

In the light of such diversity of opinions concerning the effect of a syphilitic infection upon an already existing tuberculosis it is highly desirable, as stated by Knight, that careful observations of such cases be kept and published. My personal experience includes only one other instance of the association of pulmonary tuberculosis with secondary syphilis. This was in a young man whom I saw a few times some eight years back. He had pulmonary tuberculosis in a state of rapid softening which he declared had developed subsequent to his having contracted syphilis six months prior to my examination. His pulmonary disease made rapid progress and when I last saw him it was evident that the combination of the two diseases was surely hastening his end.

In connection with this subject of the complications of chronic pulmonary tuberculosis may be mentioned the interesting and rare association of *malignant disease* and consumption. March 16th, 1904, a man of fifty was admitted to Cook County Consumptive Hospital with signs and symptoms of pulmonary tuberculosis. For ten years he had been a resident of lodging houses and had been addicted to the use of large quantities of alcohol. At the age of twenty-five he had contracted a chancre. For a number of months prior to admission he had been coughing and expectorating freely and had lost forty pounds in weight.

Physical examination disclosed dullness, bronchial breathing and fine bubbling râles in the left upper lobe, stiff arteries with hypertrophy of left ventricle and a mitral systolic murmur. The ankles were slightly œdematous. During the following six weeks he ran an irregular moderately high temperature and emaciated perceptibly. Death took place May 2, 1904.

To omit details of the autopsy it may be stated that the *post mortem* findings were: a small cavity surrounded with tuberculous nodules at the apex of the left lung and many nodules scattered throughout its base as well as old fibrous adhesions along its anterior margin; a single small area of tuberculous broncho-pneumonia in the apex of the right lung with another secondary nodule in the middle lobe; chronic arteriosclerosis with chr myocarditis and sclerosis of the mitral valve; chronic interstitial neph but more than all a cauliflower mass involving the lesser curvature of stomach and retroperitoneal lymph nodes with metastatic nodules in l and kidneys. Upon microscopic examination this mass was found to carcinoma. The cancer had produced no symptoms during life, the clin picture having been that of consumption.

CHAPTER XXVII

PULMONARY TUBERCULOSIS—*Continued*

Diagnosis.—This may or may not be a matter of difficulty depending upon the stage of the disease. When the process has become advanced, its recognition is usually easy, the diagnosis being possible in some cases by mere inspection of the patient without physical exploration of the chest or examination of the sputa. In the early stage diagnosis is not always nor even usually easy. At this period of the disease, when, from the therapeutic standpoint, so much depends on its early detection, the diagnosis may require great skill and experience, and it may be said that the determination of the true nature of the disease in its incipiency is sometimes impossible until after prolonged observation and repeated examinations.

It is because of this difficulty as well as of lack of diagnostic acumen on the part of the physician that the complaint is so often unrecognized until too late to institute proper means for its cure. On this account the early diagnosis of pulmonary tuberculosis will be given special consideration in the hope of bringing out some points of extra importance.

Diagnosis in Latent and Incipient Stage.—Although the detection of the specific bacillus in the expectoration affords the only absolutely reliable means of diagnosis in the stage of development, still there are so many cases in which expectoration is wanting at this time or fails to contain the bacilli, that one is compelled to rely upon other means. Consequently the physician must inquire carefully into the family and personal history, study attentively the various symptoms, elicit the physical signs and then compare these with the symptoms, to see in how far they agree or what may be their relation to each other.

Only in this way can the practitioner arrive at a full and intelligent understanding of his case, for, comprehensively speaking, diagnosis includes something more than mere recognition of the nature of the malady. It is not enough to say an individual has pulmonary tuberculosis; one should know in what stage the disease is, how much of the lung is affected, etc., since otherwise an intelligent view of the case cannot be had and the probable prognosis cannot be made.

Family History.—Formerly held to be important from the view of supposed hereditary predisposition, this is now regarded from another standpoint, namely that of exposure to infection. Accordingly, inquiry must be carefully made to ascertain not only whether any member of the immediate family or a relative may have had consumption, but whether, if so, the patient may have been exposed by residence in the same house with said individual. Consequently such a link of evidence is highly important since the fact of tuberculosis in the family increases the weight of other evidence pointing to its existence in the patient.

Personal History.—Minute inquiry must be instituted regarding the previous state of health, antecedent illness attended with cough and in particular pleurisy, enlarged cervical glands, hæmoptysis, etc., since, in many cases, facts may be brought out which indicate the beginning of infection months or even years earlier. Such inquiry into the personal history must also include the search after symptoms which, too trivial to attract the patient's notice, may yet be of aid in the correct interpretation of the condition. Aside from expectoration containing bacilli there are no symptoms in incipient pulmonary tuberculosis which, independent of anything else, are sufficient for a positive diagnosis in this early stage. Some of them are, nevertheless, of significance and these will now be considered in detail.

Loss of Weight.—This is one of the earliest of all premonitory symptoms. It may not be pronounced but is progressive and is associated with decrease of strength, pallor, flaccidity of the musculature, and in some cases with intellectual weakness and a degree of bodily feebleness out of all proportion to growth, occupation or any other conditions likely to produce fatigue. In short, the symptoms are those of neurasthenia.

Relation of Weight to Height.—This is also important. Normally there should be twenty-five pounds in the adult male and twenty-three in the female to each foot of height as the lowest standard of weight in health. Consequently, if the relation of weight to height falls below this standard, it should be given due consideration in arriving at a diagnosis.

Anorexia and Dyspepsia.—Failure of appetite is in most instances a noticeable feature and may be absolutely intractable to treatment. It may be free from all dyspeptic symptoms and when present in a suspected case furnishes evidence in favor of tuberculosis.

The dyspepsia of latent or incipient disease is seen in several forms (Mariani) of which the simplest is a mere functional inactivity of digestion due to catarrh. It is usually associated with loss of appetite. Another form is seen in girls who are anæmic and who present signs of chlorosis. The appetite may be capricious or perverted; eating often produces a painful sense of weight and fullness and sometimes epigastric pain. The function of the stomach is normal, however, and there may be a slight hyperchlorhydria.

The third form of indigestion which, according to Mariani, should excite a suspicion of incipient tuberculosis in girls when not traceable to any other demonstrable cause is that associated with hyperchlorhydria. This type which has been described by Marfan is distinguished by gastralgia pyrosis, and an increased appetite. The taking of food is apt to bring on a fit of coughing and this may be followed by vomiting. Marfan declares that this form of hyperacid dyspepsia may precede the development of pulmonary tuberculosis by many months and even a year. Dyspepsia of these types should arouse in the physician a suspicion of the possible development, if not the actual existence of pulmonary tuberculosis.

Anæmia.—The changes in the blood of the tuberculous have been dwelt on at length under Symptoms, but here may be emphasized certain features that may have a diagnostic bearing. The form of the anæmia is that of a chlorosis with this distinction, according to Hanot, from ordinary chlorosis, namely, not so great a reduction in the hæmoglobin and a slight increase of the leucocytes. Mariani does not appear to coincide in this statement, however, and it is my experience that as regards the leucocytes, at least, Hanot goes too far.

I have recently studied a case of pulmonary tuberculosis in so incipient a stage that local signs were quite indefinite. It was the pyrexia which first aroused my suspicions and in the examination of the blood it was found that in addition to a reduction of hæmoglobin to 65 per cent (Fleischl's method) and a considerably less decrease in the number of erythrocytes, there was a tendency to leucopæmia. On two separate occasions the white cells numbered but 5,000 and 4,700 respectively. There are few conditions in which leucopæmia occurs, and as these could be excluded, the low percentage of the leucocytes served to corroborate the conclusion to which I arrived by study of other findings.

Leucocytes.—In connection with the blood-changes from a diagnostic point of view must be mentioned the significance to be attached to a want of hyperleucocytosis. Arneth found in pulmonary tuberculosis a normal or low leucocyte count, which agrees with the reports of others as regards the count of white cells in incipient and early cases. If, therefore, a suspected patient who is running a temperature from 99.5° to 100.5° or possibly 101.5° F. shows a tendency to reduction rather than to an increase in the number of leucocytes, it to my mind furnishes a certain modicum of evidence in favor of tuberculosis. The suspicion is strengthened if, in addition, the neutrophiles show a disproportionate increase in the forms with one and two nuclei as reported by Arneth.

Tachycardia.—Acceleration of the pulse is so constant a feature of pulmonary tuberculosis that, in the opinion of French authors, this circumstance may be utilized in the recognition of incipient cases. A pulse-rate of 90 to 100 in the absence of fever or other demonstrable causes of tachycardia should always be regarded as a strong corroborative indication of latent or incipient tuberculosis (Mariani). Besançon has recorded cases of persistent acceleration of the heart's rate together with œdema and other signs of asystolism which were caused by pressure on the vagus by a mass of enlarged bronchial glands. Such a circumstance is interesting but can rarely have a bearing on the recognition of pulmonary tuberculosis.

A further noteworthy characteristic of the pulse emphasized by Faisans is its instability, i. e., the readiness with which it grows in frequency upon slight cause. This is seen particularly in persons who are erethistic as well as tuberculous. Finally the pulse fails to respond to the usual influence of posture as in health. This is evinced in particular by its frequency being maintained in the recumbent position. Accordingly, it is such features as these which should be observed in estimating the diagnostic value of tachycardia.

Lowness of Pulse-tension.—Before leaving the subject of the pulse in its relation to the recognition of latent and incipient tuberculosis I must mention the decreased tension of the pulse. Various observers have commented on this quality and it is one on which I have laid much stress in early diagnosis. Not only is blood-pressure lowered as determined by the sphygmomanometer, but in many cases, particularly when tachycardia is pronounced, the finger appreciates a striking feebleness and compressibility of the pulse. This feature taken in connection with other symptoms may, therefore, prove of aid in the interpretation of a doubtful case.

Dilatation of the Pupils.—Rampoldi originally studied the state of the pupils in pulmonary disease and Commini gave it especial attention in cases of tuberculosis. He observed mydriasis of one eye and usually on the side corresponding to the areas of greatest change. Destré following in the line of their observations has studied the state of the pupils as an aid in early diagnosis and found unilateral dilatation in 97 out of 100 cases. This mydriasis may be permanent or transient and is attributed to reflex irritation of the sympathetic by way of the vagus and inferior cervical ganglion in consequence of pressure by enlarged bronchial glands.

Turban, Amat, Souques and others have confirmed Destré's observations, and the last-mentioned observer in three cases of right-apex disease noted myosis, narrowing of the palpebral opening and diminution with retraction of the eyeball. These effects were attributed by him to destruction of the iridodilator fibers of the sympathetic in its passage through the superior portion of pleural sac in consequence of fibrous thickening of this membrane.

These various abnormalities in the state of the pupils are highly interesting and may, in some instances, serve to throw light on the existence of an early tuberculosis.

Neuralgias.—So many are the causes of neuralgia in anæmic individuals that these pains would scarcely be thought of significance in the recognition of the malady now under consideration. Nevertheless, considerable diagnostic importance is to be attached to neuralgias, especially intercostal neuralgia situated in the upper thoracic zone, namely in the infraclavicular region and behind the shoulder. Persistent pain in these situations and independent of any other demonstrable cause may, in suspected patients, prove of confirmatory value.

Erethism.—This is an unstable, excitable state of the nervous and circulatory system, which is not unfrequently seen in young persons in whom pulmonary tuberculosis is developing. I have seen it in both sexes but rather more frequently, I think, in young women. They are in some instances noticeably anæmic, but in all there is usually the history of their having always been "very nervous."

I encountered it recently in a tuberculous woman of thirty-six who was very thin, but in whom the signs of disease were not marked. Her family stated that she was very nervous and apt to faint when emotionally disturbed. Slight diagnostic value, therefore, should be attached to erethism or nervo-vascular excitability of the sort just mentioned.

Temperature.—No single symptom is so valuable as the body-temperature. Although it is undoubtedly true that cases of incipient tuberculosis are sometimes encountered which present a normal or slightly subnormal body-heat, still the rule is for a developing phthisis to be declared by slight fever.

The degree of febrile reaction is not high being in the great majority of cases from 99.4° to 100.5° F. This remark applies to ambulatory cases such as generally consult the physician in his office. Acute tuberculosis of the pneumonic type is not here included since in this form not only is fever high and pronounced, but also there is usually no difficulty in the determination of a grave pulmonary disease.

In any suspected case of incipient lung mischief the temperature should be recorded several times daily. It is my custom to have it taken and written down every three hours beginning at 8 or 9 A. M. and continuing so to do, until bedtime. This is highly important in those cases in which a conclusive diagnosis cannot be made at the first chest-examination. If the individual is suffering from incipient pulmonary tuberculosis he will generally be found to display the slight but significant elevation of temperature above mentioned, some time during the day, especially in the afternoon or evening, and with great regularity.

An irregular intermitting or even distinctly remitting type of fever does not belong to incipient tuberculosis and suggests either some other cause or some complication. On this account it is highly *serviceable to make a leucocyte count.* If the condition is tuberculosis there will be found either no increase in the number of white cells or an actual leucopæmia, whereas if the fever be due to pus infection there will usually be a leucocytosis although this may be very moderate. More will be said on this subject in the consideration of the differential diagnosis.

Another highly valuable point to be considered is the effect on the body-temperature produced by physical exercise. Slight elevation of temperature is probably always caused by prolonged exertion, as walking, yet I agree with Penzoldt and Meissen in the opinion that if the temperature be found after a walk of an hour to have risen to 100.4° F. or more, it may be regarded as very suspicious of pulmonary tuberculosis, especially if it be associated with disproportionate fatigue and other doubtful signs of incipient disease. The point is, that such an elevation of temperature is highly important in diagnosis, only when the individual is known to have practically a normal temperature directly prior to his walk. If the thermometer be held in the mouth, which by the way is not to be recommended in so important a matter as the one now being considered, the record will be, it must be remembered, a degree lower than in the rectum.

In all cases therefore of doubtful diagnosis an opinion should be withheld until after the daily temperature has been measured for at least a week in the manner described. Thus taken it will be found of the greatest aid arriving at a clear understanding of the true nature of the case.

Lastly, it must be borne in mind that in tuberculous women a moderate e reaction takes place at the time of the menses. Such an increase of body-temperature may be taken, therefore, as a strong indication of latent or incipient tuberculosis.

Cough.—This is one of the earliest symptoms to which diagnostic significance may be attached. It varies much, however, in different cases. It is generally short and dry and may sometimes be so slight as to be denied altogether or admitted to occur only in the early morning. Although having been of the opinion that some degree of cough is never really wanting, I have recently observed a case in which this symptom did not exist. It may be very infrequent as well as trifling or it may in rare instances prove a most troublesome feature. Cough is of value in the early diagnosis but only when associated with other confirmatory symptoms and physical signs.

Expectoration is absent in the latent and often in incipient cases and when present is scanty and mucous, catarrhal as it is called. Its value in diagnosis lies in the possibility of its containing tubercle bacilli. Consequently, the presence or absence of expectoration should be ascertained and one or more specimens of sputum should be demanded for microscopic examination.

If bacilli are detected the diagnosis is settled at once, but unfortunately this is frequently not the case in the incipient stage. In such an event one should not content himself with a single examination but must carefully inspect daily samples of sputum over a considerable period, on each occasion searching assiduously through a number of slides.

Method of staining and examining sputa for tubercle bacilli. This is based on the fact that the bacillus when once properly stained tenaciously retains the stain even when subjected to the action of a mineral acid, whereas all other bacteria that happen to be also present become decolorized.

Morning sputum is best, since most likely to contain the germ. It is recommended that one of the firm grayish masses be selected and by means of a platinum wire or suitable forceps be laid on the surface of the cover-glass, crushed and then spread out in a thin uniform layer. After this has been fixed by heat in the ordinary way it is then stained as follows:

(1) Stain the entire surface of the cover-glass with a carbol-fuchsine solution (ten parts of a saturated alcoholic solution of fuchsine in ninety parts of a 5-per-cent solution of carbolic acid, the so-called Ziehl-Neelson solution) and then steam gently over a Bunsen flame. (2) Wash in distilled water. (3) Decolorize with a 30-per-cent aqueous solution of strong nitric acid until the red color disappears, but do not allow the acid to remain in contact with the preparation for more than a few seconds lest the germs themselves be deprived of the stain. (4) Wash thoroughly in water as before. (5) Wash for thirty seconds in 95-per-cent alcohol. (6) Stain with solution of methylene-blue. (7) Wash in water, mount and examine.

The above method may be shortened one step by the use of Gabb solution of methylene-blue which is acid, as follows, (methylene-blue t parts, sulphuric acid twenty-five parts and water seventy-five parts) a serves to decolorize other bacteria while at the same time staining w blue all but the tubercle bacilli. The method just described is simple rapid and with a little practice will enable anyone to recognize the ba which are seen as red slightly curved rods such as have been shown in Plate VII.

Sedimentation of the sputum is resorted to for the purpose of enabling one to discover tubercle bacilli when present in small numbers. A ready means of precipitating the solid portion of the sputum is recommended as follows: Either by means of boiling water or steam the sputum is heated in a test-tube and then allowed to stand for fifteen minutes. This coagulates the cells and albuminous ingredients of the sputum which sink to the bottom as a dense sediment. After having poured off the supernatant fluid, the precipitate is stained and examined in the manner given above.

As has been stated already, the recognition of the specific bacillus is the only absolute means of diagnosis if one speaks in a strictly scientific sense. Unfortunately expectoration is not always to be had in cases of latent and incipient pulmonary tuberculosis and it is in these very cases that an early diagnosis is desirable. Consequently the physician must rely not upon laboratory but upon clinical diagnosis, such as I am endeavoring to bring out under history, symptoms and physical signs.

Hæmoptysis.—The consideration of the symptomatic diagnosis of incipient tuberculosis would not be complete without a few words devoted to this symptom. Expectoration of blood is always suggestive of the presence of tubercles in the lungs, especially when it occurs without previous indications of disease. Although it may not necessarily arise from tuberculosis it does, nevertheless, owe its origin to this affection in such an overwhelming proportion of cases that when more obvious causes are not discoverable hæmoptysis may be considered of immense diagnostic value even in the absence of demonstrable pulmonary lesions. For further particulars on this subject the reader is referred to the chapter dealing with Pulmonary Hæmorrhage.

Physical Signs of Diagnostic Import.—In few departments of clinical diagnosis is greater expertness required than in the detection of those trifling deviations from the norm, as they might seem to the tyro, yet which are of such significance to the skilled diagnostician. The first essential, therefore, is a knowledge of normal differences and the second is the ability to determine the relative value of physical signs when compared with or set over against certain symptoms.

I would especially caution the reader against mistaking the changes in pulmonary resonance and in breath-sounds caused by deformities of the thorax for those produced by disease of the pulmonary parenchyma. Spinal scoliosis, even when slight, may, and often does, occasion perceptible impairment of the lung that is impinged upon by the spinal curvature, and may on this account occasion difficulty.

The greatest perplexity arises, however, when an individual with scoliosis or kyphoscoliosis is, as a matter of fact, in the very earliest stages of pulmonary tuberculosis and the changes in the lung are on the side toward which the spinal column deviates. In such a case very great judgment and discrimination are necessary if one is correctly to interpret the clinical findings.

Changes to be sought for by (a) Inspection.—These are (1) *slight wasting of the scapulo-humeral or shoulder-girdle muscles.* This is one of the earliest signs and, in my experience, is rarely missed if carefully sought for. I am usually able to detect it most plainly in the muscles covering the supraspinous and infraspinous fossæ of the scapula.

The wasting of these muscles can be perceived by looking down on them from behind and one side or by passing the hands gently over them on the two sides simultaneously. If the tuberculous deposits are unilateral and the muscular wasting exists only or is more marked on the same side, the atrophy can usually be readily distinguished and furnishes a valuable early sign of incipient tuberculosis.

(2) *Flattening of the chest in the infraclavicular region on the affected side* is another sign perceptible by inspection. This, in early cases, is the result of the muscular atrophy which involves the pectorals as well as the muscles on the posterior aspect of the shoulder. This flattening is associated with more or less sinking of the supraclavicular region so that the collar bone stands out more prominently than does its fellow and the entire apex looks somewhat shrunken.

(3) In early cases there is usually observable on very careful inspection some difference in the two apices as regards their respiratory movement. This is shown by *retarded expansion of the affected apex* on inspiration. Considerable skill is required to appreciate this difference in cases of limited tuberculous infiltration, since, if the patient is told to take a deep breath, he is apt to call into play the auxiliary muscles of respiration which then lift rather than expand the chest and thus tend to obscure the very thing the physician is desirous of seeing. Consequently it is better attentively to inspect the respiratory play during quiet or but slightly deepened respiration.

(4) *Diminished excursion-movement of the diaphragm* on the affected side is another early sign insisted upon by Francis H. Williams and others who are skilled in the use of the fluoroscope for the diagnosis of incipient cases. It is caused by a loss of elasticity of the lung the same as is the restricted expansion of the apex, or by pleuritic adhesions which may likewise exist at the summit of the lung.

If the physician has not access to or skill in the use of the X-ray he may be able to detect this defective play of the diaphragm by looking for *Litten's shadow-sign,* care being taken to compare the two halves of the thorax, since in some individuals this shadow-sign is naturally ill-defined.

If a person lie with uncovered chest upon a couch or table in a good light and the physician standing at his feet watch attentively the lower portion of his chest during quiet respiration, a distinct shadow can usually be seen to move downward during inspiration and upward during expiration on that anterior inferior portion of the chest-surface at either side corresponding to the situation of the diaphragm.

This is the shadow caused by the movement of the diaphragm original described by Litten. In health this shadow has a play of from one to t inches and when well marked is a good index of the expansion of the lun It is not well seen in women with large mammary development and restricted or abolished by pleuritic adhesions or by any other disease which interferes with the respiratory play of the lungs. Consequently, the loss or unmistakable diminution of this shadow on one side furnishes information which, taken in connection with other obscure physical signs may aid materially in the recognition of incipient tuberculosis.

In utilizing the knowledge to be gained by inspection in these early cases one must bear in mind that as there are wide differences in the extent of the tuberculous deposit and in the length of time during which tubercles have been developing, so there are corresponding differences in the distinctness of the early clinical manifestations. In one case the physical signs may be quite distinct yet slight, whereas in another they are indistinct or even wanting altogether. *It is the detection of very slight differences, therefore, which is important.*

(*b*) *Palpation.*—I have rarely found this of material aid in the diagnosis of latent and incipient cases. Theoretically, the consolidation of pulmonary parenchyma occasioned by the growth of tubercles ought to facilitate the transmission of vocal fremitus and this increase of vibration ought to be available for diagnosis. As a matter of fact, such is sometimes the case and the hand is able to appreciate more fremitus over one apex than over the other.

There are other cases, however, in which the pectoral fremitus is diminished rather than intensified over an apex which subsequent percussion shows to be distinctly impaired. It has happened to me more than once that, having palpated the chest as is my wont and having detected greater vocal vibration on one side and hence assumed that that must be the affected apex, I have been surprised by finding it the more resonant of the two.

In such a case I presume the apparently incongruous findings are due either to old pleuritic thickening or to small caseating areas of tuberculous broncho-pneumonia which, by obliterating whole groups of lobules, occasion loss of resonance and in some way interfere with the conduction to the surface of the vibrations in the column of air produced by spoken words.

The real value of palpation lies in the detection of augmented pectoral fremitus over a small circumscribed area at one apex, e. g., in the suprascapular region (see Fig. 52) where occurs the primary tuberculous deposit. Such a finding would indicate some localized condition favoring better conduction; and what more likely in a case of suspected tuberculosis than a patch of broncho-pneumonia offering suitable conditions for its detection by this method! Nevertheless, it is my belief that when such a patch is discoverable by palpation, it is also detectible by percussion and with greater certainty.

(*c*) *Percussion.*—So much depends upon the skill of the examiner in practising this mode of chest-examination that two or more men may draw different conclusions from percussion of a case at the same sitting. Moreover, the situation and extent of the lesion are of such vital importance as to make the results of percussion uncertain. Theoretically a lung that is the seat of tubercle should yield more or less loss of percussion-resonance, but in actual practice we do not always find demonstrable impai percussion-note. This may be attributable either to the small size of the tuberculous focus or to its separation from the surface by a sufficiently thick layer of healthy parenchyma to conceal the tuberculous mass beneath.

For the foregoing reasons I recommend that in any doubtful case, percussion be performed alternately by a light and firm lingering or legato stroke while at the same time the pleximeter finger is pressed firmly and evenly against the surface of the chest. I am convinced also that the fingers are preferable to the use of hammer and plessimeter of whatever material, since they enable one to appreciate an increased sense of resistance and this is a positive aid in detecting small consolidated areas.

The percussion-stroke does not need to be heavy and sledgehammer-like. Indeed such is usually a mistake in these early cases. Let it be gentle and of varying degrees of strength since by so doing one may detect foci at a depth, or pleuritic thickening at the periphery. Even when performed in this manner and with all the skill of which the physician is capable he may fail to detect any difference in the resonance of the two apices. Should slight dullness be detected, it is more likely to be at the apex behind than in front for reasons previously stated under Physical Signs. In most cases when the suprascapular region is impaired it is found that resonance is also impaired above the clavicle and that the apex is slightly retracted.

Tuberculous disease causes retraction of the lung at the site of the lesion, and hence an appreciable shrinkage of the affected apex is one of the *earliest percussion evidences of incipient phthisis.* It will be noted that I said one of the earliest percussion evidences of incipient phthisis. This was done advisedly because I agree with Granger and others in the opinion that auscultation is a more delicate means of recognizing slight circumscribed changes than is percussion.

Impairment of resonance at one apex may be caused by other conditions than tuberculosis, e. g., kyphoscoliosis; yet it may be accepted as a general proposition that dullness in this situation points to the disease we are now considering. In cases not due to tuberculosis, however, there is apt to be a general loss of resonance not merely along the inner border of the lung but to some extent throughout the entire upper lobe. Whereas in tuberculosis the earliest changes are circumscribed as was shown in considering the morbid anatomy. Consequently, if the physician can detect slight dullness over a limited area behind and not over the greater portion of the lobe, he may, if other data corroborate, set it down as indicative of latent or incipient tuberculosis.

(*d*) *Auscultation.*—Herein lies the most delicate means of detecting incipient disease but it presupposes accuracy of hearing and perfect familiarity with normal breath-sounds. These conditions are essential on the par the examiner since he is called on in this class of cases to appreciate yet significant modifications of the respiratory murmur.

Bearing in mind the fact that the primary site of tuberculous de is in the posterior portion of the apex, about an inch from its summi examiner should direct special attention to the suprascapular and n the infraclavicular region, as is only too often done. If disease exist breath-sounds at this point are likely to present either of two deviations from the soft vesicular murmur of health. The more frequent of these is ***roughness of the inspirium.*** This is the characteristic insisted upon by Granger and other French writers as furnishing the earliest appreciable evidence of latent or incipient tuberculosis.

My experience is in full accord with this statement. In addition there is usually a lengthening of the expirium which may at the same time possess a somewhat sharpened quality. Lengthening of expiration is, however, of less significance than is roughness of inspiration. This latter quality often is found to stand out with marked distinctness when comparison is made between the sounds in this area and those of the parts roundabout or on the opposite side.

In a smaller proportion of cases the breath-sounds are enfeebled or, as the Germans say, indeterminate. This peculiarity is also highly suggestive of tubercle but does not, as it seems to me, possess the same value as does the roughness of the inspiratory sound.

Finally the voice-sounds must also be carefully studied but they do not always furnish as much information as do the breath-sounds. When tuberculous infiltration exists the voice is likely to be exaggerated (bronchophonic) and the whisper is more than usually clear and sharply defined.

As the disease advances to the production of signs and symptoms marking the stage of activity the areas of secondary involvement appear and diagnosis becomes easier. There is now roughness of breathing in front which may really be classed as broncho-vesicular. But by the time auscultatory evidence of disease appears in front there are apt to be adventitious sounds behind which render error of diagnosis scarcely possible.

These are a collection of fine clicks or crepitant râles which are heard over the upper portion of the suprascapular fossa. They are very like the crepitant râles of acute pneumonia but are not so sharply crackling. As might be expected these râles are heard behind at an earlier period and more distinctly than elsewhere for the reason that the disease advances more rapidly posteriorly than in front. They are brought out more clearly by deep inspiration and by cough, especially if the cough be made to follow expiration immediately, that is, before another inspiration is taken.

Under such conditions the râles or *signs of moisture* as they are often called, are so characteristic that they cannot well be confounded with any other adventitious sounds. They are not pathognomonic but are very diagnostic when combined with other data described above.

Isolated subcrepitant râles over an apex high up may sometimes be simulated by indistinct crackling sounds generated in the throat by the act of swallowing and hence patients with doubtful signs should be cautioned against swallowing during auscultation of the apices. With ordinary care, however, mistakes of this kind can be avoided. A less frequent râle is a fine sibilus over the affected apex. It may mislead one into the belief that the case is an acute bronchitis but if the râle is posterior or confined to one apex, it has the same significance as has crepitus in this situation.

Summary of Diagnostic Data in Incipient Cases.—(*a*) History of possible infection by residence in the same house with a tuberculous relative. (*b*) History of previous pleurisy, etc. (*c*) Gradual loss of weight not attributable to other disease or ascertainable cause. (*d*) Gradual decline of strength with or without loss of appetite. (*e*) An anæmic or ashen gray color, often with a bright eye. (*f*) Daily temperature from 99.4° to 100.4° F. being generally most marked and constant in the afternoon or early evening. (*g*) Pseudo-chlorosis with normal or diminished number of the leucocytes and Arneth's changes in the neutrophile elements. (*h*) Slight dry cough or, if there is expectoration, this may be with or without bacilli. (*i*) Frequent, low-tension pulse. (*j*) Hæmoptysis days, weeks or possibly months before. (*k*) Slight atrophy of the shoulder-girdle muscles. (*l*) Slight flattening or depression of one apex. (*m*) Loss of Litten's shadow-sign on one side. (*n*) Slight increase of pectoral fremitus over a limited area at one apex generally behind. (*o*) Slight impairment of resonance at one apex usually more apparent behind over a circumscribed area corresponding in site to the primary focus of disease. (*p*) Roughened inspiration in the same situation or indeterminate or broncho-vesicular or even feeble bronchial breathing at one apex, especially behind. (*q*) Sibilus or fine crepitant râles at an apex, very limited in extent and brought out more clearly by deep inspiration and by cough.

It would be an error to suppose that all of the points are present in every case of suspected tuberculosis. The most of them are likely to be, and yet one is frequently required to make a diagnosis in a case showing only slight decrease in weight, debility and cough. Physical signs may be inappreciable or so vague as to be of very little aid. In such an event the study of the temperature and the blood-count must be relied on, and if these agree with what one would expect to find in incipient tuberculous infection and if they are associated with changes at one apex of such indefinite character as to leave one in doubt when depending on these last alone; if, I repeat, there be such a conjunction of symptoms and uncertain signs in a suspected case, the diagnosis should be made inferentially at least.

It is a serious matter to make a wrong diagnosis in either direction, yet, as regards the future welfare of the individual, it is better incorrectly to state incipient tuberculosis to be present, than to overlook it. Still one should never rest content with uncertainty, and when one of the not infrequent cases arises in which one is dubious, even after the most careful and painstaking study, only two steps are open to him. Either he must keep the individual under close observation until by repeated and frequent examinations he can arrive at a positive opinion, or he must resort to the tuberculine test.

The Use of Koch's Tuberculin for Diagnostic Purposes.—This agent has now been employed in so many cases in both man and animals that its use can no longer be considered experimental. Two queries arise, however, in this connection, namely, is it safe to use tuberculin for this purpose and does the characteristic reaction necessarily denote pulmonary tuberculosis?

In answer to the first question it may be replied that its safety depends on the size of the dose and the manner of its administration. Pulmonary hemorrhage and acute tuberculous broncho-pneumonia have been known to follow its injection in a few instances, yet such is their rarity that they may be left out of account, provided proper precautions are observed.

The initial dose is from 1 to 3 milligrams, but by some clinicians a single milligram is not considered sufficient to produce reaction in most cases of latent tuberculosis. Should the initial dose, whatever this may be, fail to produce the characteristic reaction, a second injection of double amount may be given after a lapse of three days. Should this second dose be also negative, then after another interval of three days a third injection of six to ten mg. may be administered. Should this last-mentioned dose of 10 mg. prove likewise fruitless the case may be dismissed as probably not tuberculous.

In most instances, if tuberculosis be present, reaction will follow before so large an amount as 10 mg. has been reached. It is better to begin with the minimum dose and very gradually work up to the limit even if more than three injections are required than to do harm by too high an initial dose or too rapid an increase.

The temperature should be recorded several times a day for at least three days prior to the test, and as the reaction is likely to occur at the end of about twelve hours it is best to make the injection in the late evening so that the results may be observed on the following day.

Reaction is evinced by rise of temperature of at least 2° F., aching of the extremities and back and in some cases by nausea and vomiting. The more marked the reaction, the more intense are the constitutional symptoms. In some cases examination of the lungs discloses fine moist râles at the seat of lesion. The constitutional disturbance usually subsides in from twenty-four to forty-eight hours.

The second query concerning significance of the reaction is also pertinent. As a matter of fact tuberculosis is not the only disease, e. g., syphilis, which favors the production of symptoms that indicate reaction. Nevertheless, such is their rarity that a reaction may be regarded as denoting tuberculous disease. Herein lies the difficulty as respects latent or incipient lesions in the lungs. The characteristic reaction may ensue and yet the disease be situated in some other structure as, e. g., bronchial or mesenteric glands.

If an individual complaining of debility, with or without certain chest symptoms, as cough, displays reaction to tuberculin, he is probably tuberculous; and if a careful chest-examination has revealed signs of doubtful significance before the test, suspicion of pulmonary tuberculosis becomes a certainty in the light of the reaction.

My personal experience with this means of diagnosis has not been great, for I can now recall very few cases in which I have not been able to arrive at a definite diagnosis by recourse to the various other means recommended; as record of the daily temperature for a week, examination of the blood and a second or third chest-examination. I do not lay any claim to greater skill than can be acquired by any practitioner of twenty years' experience in this class of cases. I mention this only to encourage the young physician to painstaking investigation.

Early Diagnosis by Means of the X-ray.—With this means of diagnosis I have had absolutely no experience. Consequently what I have to say is based upon reports given me by Dr. C. L. Minor and on the writings of Dr. Francis H. Williams, Aschard, Mayhew and Solly. The testimony of these experts is, that in the X-ray lies a very reliable means of diagnosis before this is possible by ordinary physical exploration of the chest. Williams states that the fluorescent screen has disclosed the presence of tuberculous foci in the lungs in cases in which it had not even been suspected.

Pulmonary disease of this nature is revealed by darkening of the lung in the affected area and by restriction or complete abolition of the excursion movements of the diaphragm on the same side. In some cases the heart may be seen to be displaced somewhat toward the diseased lung. The darkening of the lung observed by the fluorescent screen is due to its increased density, and hence the degree and extent of the darkening is a measure of the extent and character of the changes produced.

There may be a comparatively uniform diminution in clearness when foci are disseminated or there may be circumscribed areas of darkening at the apex. Some care and skill are necessary if one is to obtain trustworthy results, since to perceive definite differences between the two apices one must inspect the lung with varying degrees of intensity in the light.

Occasionally the radiograph will bring out more clearly, foci which were not perceived by aid of the screen, but as a rule the screen alone is sufficient. This device is of especial aid also in enabling the physician to judge of the results of treatment and to determine whether the disease is advancing or decreasing.

Valuable, however, as the X-ray is as a means for early diagnosis Williams very prudently advises against relying on it exclusively and urges that it be used in conjunction with other methods, as physical examination and the tuberculin test.

There are other conditions than tuberculosis which may occasion an obscuration of the lung. Among them are pneumonia and active or passive congestion, a very thick chest-wall, tumors, syphilis, etc. Consequently, it is necessary to estimate the disclosures of the X-ray in the light of the anamnesis and general symptoms as well as the location and extent of the darkening perceived by means of the fluorescent screen.

With respect to the use of the X-ray for diagnostic purposes Aschard says the examination must be made at various angles, including the oblique, and over a limited area at a time by means of a lead shield. If a radiograph is to be obtained the picture should be taken during the suspension of breathing by what he terms a "snap-shot method." The X-ray examinat
he thinks, not so much the tubercles themselves as the changes the
A spotted or dappled appearance of the lung is seen only in adva
while in the early stage there is only a darkening or relative opa
apex which necessitates care for its correct interpretation. On
therefore, Aschard believes the X-ray reveals the advent of tubercl
certainty than it does the existence of old lesions, but is, on this ac

the less valuable to the clinician since even in such conditions a diagnosis is rendered possible which, from a practical standpoint, may be considered early. Mayhew relies upon the skiagraph to the exclusion of the fluoroscope and has exhibited numerous plates which prove the usefulness of this means of diagnosis in both early and late cases. Solly lays particular stress on the value of the skiagraph for the exclusion of tuberculosis in doubtful cases as well as for the detection of disseminated foci not open to detection by physical examination of the chest. It may here be added that in the X-ray lies a means of detecting and outlining cavities.

Spirometry.—The detection of pulmonary tuberculosis is of inestimable importance in its period of development, i. e., before it has begun to occasion appreciable influence on the general health, or when there is doubt whether such symptoms as exist are to be attributed to an incipient manifestation of disease. Its diagnosis at this time is so difficult, moreover, that ingenious minds have been and still are active in the invention of mechanical appliances that may prove of aid in this matter. The general practitioner may not be able to command these and might not be able to use them skillfully if he possessed them. Moreover in my opinion the most of them are not so practical as to make them of material assistance, but inasmuch as they have either received the indorsement of clinicians or are now attracting their attention, they will be described.

The spirometer is an apparatus for the measurement of the respiratory capacity and in its best form consists of a tank or vessel which is inverted and submerged in a water-bath. By means of a rubber tube the exhaled breath may be conducted to the interior of this chamber which then rises above the surface of the water to a height depending upon the quantity of air thus exhaled. A manometer connected with this movable chamber registers the amount of air expired.

In a healthy individual there is a constant and definite relation between the stature and the respiratory capacity. For a man this is 20 ccm. of air to each cm. of height while in an adult woman this ratio is 17 to 1. If the lungs are diseased respiratory capacity is reduced, therefore a reduction of the normal ratio is considered suspicious of pulmonary tuberculosis when no other gross pathological lesions are determinable. Spirometry is open to the objection, however, that practice and skill in the use of the apparatus, as well as other factors outside of the lungs have much to do with the results. For this reason I long ago abandoned the use of this instrument. It may be of service, nevertheless, by enabling the phthisiotherapist to determine the results of treatment in a given case.

Pneumography.—This is a method of obtaining a graphic representation or tracing of the respiratory movements, on which Hirtz and Brouardel are said to lay particular stress. They find that in normal respiration the tracing is made up of four lines as follows: the first is a line of ascent which corresponds to inspiration, the second corresponds to the acme or summit of the respiratory curve, the third or descending line to expiration and the fourth to the period of repose or apnœa. In the early stage of pulmonary tuberculosis these lines are reduced to three in consequence of a lengthening of the line of expiratory descent and the obliteration of the fourth line or line of pulmonary collapse.

Aschard in his report on New Methods of Diagnosis before the International Congress on Tuberculosis, Paris, 1905, is authority for the statement that the conclusions of Hirtz and Brouardel are controverted by the observations of Letulle and Pompilian. These investigators find the respiratory rhythm of the tuberculous so variable that no type can be determined which may be regarded as characteristic of such individuals.

Robin's Measurement of Gaseous Exchange During Respiration.—The experiments along this line made by Robin and Dinet have been referred to already in Analysis of Symptoms under the discussion of the respiratory system. According to their elaborate series of investigations there is an increased activity in the tuberculous as regards the excretion of CO_2 and the intake and absorption of oxygen. Accordingly, they believe that in the detection of such an increase of respiratory activity may be found an important aid in the early diagnosis of bodily states predisposing to pulmonary tuberculosis and also of latent and incipient disease.

As Aschard very pertinently remarks this method of studying the activity of respiratory chemistry is highly interesting and may prove of immense value to the pathologist. It can scarcely prove of practical aid to the clinician, however, in determining the presence of latent tubercles in the lungs. The apparatus is very simple but in carrying out the technique of the method very great care and patience are required. Not only must the physician spend much time in conducting the investigations in each instance, but the individual must be calm and at repose in both mind and body so as to breathe naturally and quietly—conditions which render the application of the method open to great error.

Robin's original communication on this subject to the Paris Academy is full of interest and to it the reader is referred. It was found that an augmentation of the gaseous exchange occurs in a variety of other states as typhoid fever, dyspepsia, pleurisy with effusion, ascites, diabetes, carcinoma of the stomach, hæmorrhage and chronic alcoholism. Some of these are conditions which may predispose to tuberculosis, but the determination of the fact of an increased activity of respiratory chemistry in these states does not by any means settle the question whether pulmonary tuberculosis also exists.

On the whole, therefore, the conclusion seems justified that as yet we possess no means of detecting the presence of tubercles in the lungs, prior to the outbreak of active symptoms, more reliable than is a physical examination of the chest. The one exception to this statement may be found in a tuberculin test. Yet even this does not necessarily declare that the lungs are t
tubercles; for assuming that a characteristic reaction points to tu
it does not prove it to be pulmonary. It may enable the physician t
the true value of certain indefinite or obscure pulmonary findings.
also occasion the appearance of apical râles, but in the tuberculi
itself, apart from such added evidence, does not exist a means of d
latent pulmonary tuberculosis. Therefore the physician cannot to

and judiciously study the physical signs and general symptoms as they have here been described.

Diagnosis in a Later but Still Early Stage.—In this stage the growth of tubercle is no longer restricted to the primary focus but has spread to secondary foci and led to broncho-pneumonic areas of varying size and number. The intervening parenchyma is congested, there is localized bronchitis and some of the older zones are even beginning to undergo caseation. The process may still be regarded as an unmixed tuberculosis.

The resulting constitutional disturbance is somewhat more marked than in the formative period just considered and yet is not such as to give the individual the consumptive appearance of the advanced stage. The local process is still confined to one apex but is more advanced behind, where it may have invaded the summit of the lower lobe.

Diagnosis is now sufficiently easy, as a rule, to make inexcusable the failure to recognize the condition by a careful examination. The changes perceived on inspection and palpation may or may not be more pronounced than in the incipiency of the disease, but on percussion and auscultation the signs of apical mischief are definite though still not conspicuous.

The affected apex is appreciably though not markedly dull, especially behind, while in front in the first or second interspace the note may have a wooden quality. It is imperative, therefore, that the physician examine the back, and it is my custom to begin percussion in the suprascapular region. Corresponding to the density and extent of the infiltration the breath-sounds are broncho-vesicular or bronchial and the voice bronchophonic. The breath-sounds are, moreover, accompanied or obscured by fine mucous or sibilant râles of bronchitis and often by delicate clicks in the suprascapular fossa which crepitations are brought out more numerously and loudly by cough or deepened inspiration.

The location of the affection at one apex, together with the anamnesis, attests its tuberculous nature, while the extent and distinctness of the findings show that it has passed from incipiency into the stage of confirmed pulmonary tuberculosis. Whether it is to be classed as in the first or second stage is to be determined by the symptoms as well as by the physical signs.

These have been stated in the paragraphs preceding and hence do not require repetition. If the temperature exceeds 100.5° or possibly 101° F., if the sputum is no longer mucous but contains pus and if, instead of fine crepitus or sibilus largely confined to the suprascapular fossa, the râles are found to be composed of medium-sized bubbling and clicking sounds, the infection has become mixed and although still early in the matter of time or duration cannot strictly be included in the first stage.

The following is a case in point. A gentleman of thirty-three consulted me because of a cough that had annoyed him for three or four weeks. At first it was without expectoration but for the last few days this had been considerable. He did not think he had lost much in weight or strength, yet admitted that he had been running down for several months prior to the onset of cough.

Questioned closely concerning the sputa and the presence or absence of fever, he stated that the former had become greenish yellow and of considerable amount, possibly half a teacupful daily. His wife then testified that he had a good deal of fever which the evening previous had risen to 102° F. and been ushered in by a chill and succeeded by sweating.

At the time of my examination (2 o'clock P. M.) the temperature in the mouth was 99.9° F. At the left back, from apex to level of the inferior angle of the scapula, down the interscapular region were numerous small and medium-sized bubbling râles, a few being also heard in front in the first interspace. Dullness over these areas was not intense, yet plainly evident. At the right apex resonance was impaired and breathing was harsh without moisture.

The history in this case indicated that tuberculosis had been developing for several months prior to the onset of sufficient cough to attract special notice. Then came a short period in which an examination would have revealed signs of incipient mischief. This in turn was followed by a secondary mixed infection and at the date of my examination the process had passed from the first into the second stage. Softening was setting in rapidly and would shortly become pronounced. Bacilli were found in the sputum expectorated in my office but no shreds of pulmonary tissue were discovered.

Diagnosis in the Moderately Advanced Stage or Stage of Softening.—The reader should remember that the various changes which a tuberculous lung undergoes are not sharply differentiated either pathologically or clinically and hence cases may be encountered that stand on the border line, as it were, between the first and second stages. When an individual is distinctly consumptive he is well on in his disease and diagnosis is generally easy. This is certainly true if the process is active.

To the skilled observer the presence of consumption is now stamped on every feature; in the bright eye, the hollow, often flushed cheek, contrasting sharply with the unhealthy pallor of the rest of the countenance, and the frequent loose cough and abundant purulent expectoration. The temperature range is much greater than at an earlier period, reaching one and a half, or two degrees higher than the upper limit of the fever of incipiency.

Chilliness ushers in the exacerbation of the pyrexia and distressing perspiration marks its decline. Debility, anorexia and emaciation are all more marked than during the first stage and the patient is aware of the fact that he is rapidly losing ground. Examination of the chest makes diagnosis plain and easy. The chest is thin and sunken, one apex being usually more retracted and immobile than the other. Pectoral fremitus is apt to be exaggerated over the upper portion and percussion reveals pronounced dullness over the whole of one upper lobe and possibly part of a second. As always the dullness extends to a greater distance down the back than the front and is more inten behind.

Breath-sounds are more or less distinctly bronchial and there is bronchop ony or pectoriloquy in the infraclavicular region. Râles are ususally abu dant and consist of loud large clicks even on quiet respiration while forc inspiration and cough produce numerous fine crepitations even beyond margins of distinct dullness. The perspiration often drips from the ax during the examination and the body emits a penetrating, disagreeable odor unlike that given forth in any other disease.

The conclusion to be drawn from the foregoing data is confirmed by examination of the sputa. These contain tubercle bacilli in numbers, with pus organisms, showing the mixed character of the affection, and sometimes elastic fibers or even small clumps of pulmonary tissue.

Diagnosis in the Far Advanced Stage or Stage of Cavities.—In this stage which is only an advanced degree of that of softening, diagnosis is rarely perplexing because of the more intense character of the constitutional symptoms and of the still greater extent of lung involved. Signs of disease are found in two, often in three and sometimes in four or all five lobes.

At the apex primarily invaded are signs indicating cavity and fibrosis, nature's attempts at healing, while at the base the findings are those of softening and broncho-pneumonia with pleurisy. The changes in the opposite lung are similar but less advanced. From a morbid anatomical view all the changes undergone by the disease are represented, but the clinical picture has received the stamp of advancing destruction or of an attempt at arrest according to the predominance of the changes going on in the lungs.

Signs of Cavity.—The detection of vomicæ may or may not be easy according to various conditions, as size, location, emptiness or fullness, rigidity of their walls, their ability to reflect vibrations, etc. Consequently we may meet with cases of advanced phthisis in which we at once discover one or more large cavities, or we may see others which are obviously in the last stages of the disease and yet, in spite of the foregone conclusion that excavation must have taken place, we cannot do more than infer as much.

If a consumptive is plainly in the last stage of his malady what difference does it really make if we cannot locate a cavity. Now and then, to be sure, it is important to determine the presence or situation of a vomica, e. g., in a case of severe hæmoptysis, since it has much to do with prognosis, or in the event that an operation for drainage of the cavity is to be attempted. Therefore, as remarked by Leube, determination of a cavity may rightly belong to the province of diagnosis.

As a rule, inspection and palpation are of slight if any aid in diagnosis, and one depends upon the results of percussion and auscultation in recognizing the existence of a vomica. Nevertheless, cases are now and then seen which display localized depression of the chest-wall, e. g., below one clavicle, or a circumscribed bulging on cough over the site of the excavation; or palpation may detect pronounced vocal fremitus over an area which is tympanitic so that it is plain that it cannot be induration of the lung which occasions the abnormal transmission of the vibrations. Such evidence is not conclusive however since it is possible for a large bronchiectasis to occasion such signs.

To be accessible to percussion a cavity must be of the diameter of an English walnut at least (Leube). It must be situated close to the wall of the thorax or be separated therefrom by solidified lung,—conditions requisite for transmission to the air contained in the cavity of vibrations set in motion by the percussion-stroke. Moreover the walls of the vomica must be flexible and their inner surface sufficiently smooth to reflect the waves of air striking upon them. It is the failure of all these conditions to be present in all cases which accounts for the lack of percussion-evidence so often when *a priori* one knows that a cavity must exist.

A *percussion-note of tympanitic quality* in the midst of surrounding dullness as well as *cracked-pot resonance* over a circumscribed zone furnishes presumptive evidence of the existence of a vomica and such may suffice for diagnosis when precise information is not required, as in a contemplated pneumotomy. The *bruit de pot fêlé* may be produced by a too energetic percussion-stroke on a very yielding thorax, e. g., on a baby with bronchitis or when uttering a cry. Cracked-pot sound is not pathognomonic therefore, unless it has at the same time a metallic quality, when, according to Leube, it may be regarded as indicative of a cavity. Heard over an area of circumscribed tympany in the midst of surrounding dullness it is certainly suggestive of either a true pulmonary vomica or of a bronchiectasis and its diagnostic value must be determined by other signs now to be described.

Wintrich's Change of Pitch.—This is a change in the pitch of the percussion-note produced by the alternate opening and closing of the patient's mouth. The note is higher when the mouth is open and appreciably lower when it is closed. This sign furnishes only inferential evidence of cavity since an identical phenomenon may be occasioned by percussion over a bronchial dilatation.

Gerhardt's Change of Pitch.—This is a change in the pitch of the percussion-note depending upon the position of the patient's body, it being higher in the recumbent and lower in the erect posture or the reverse. This is a very strong sign of vomica but cannot be called pathognomonic. Leube expresses the opinion, however, that when the note is found to be thus influenced by position it is proof of the existence of a cavity. These modifications of the pitch are not often observed and hence not available in most cases.

The Interrupted Wintrich's Change of Pitch.—This term is applied to the phenomenon when its production depends upon the opened or closed mouth, as well as the position of the body, that is, when the percussion-note is found to vary in the recumbent but not the erect posture or the reverse. This is therefore a combination of both Wintrich's and Gerhardt's signs.

Inasmuch as Wintrich's sign depends for its production upon the free communication of the air in the cavity with that in the mouth through a good-sized intervening bronchus, it is evident that when change of pitch is elicited in one and not another position it is because, under the effect of gravitation, the contents of the cavity may or may not, according to circumstances, interfere with the communication of air mentioned. Hence it is that the interrupted change of pitch furnishes still stronger evidence of the existence of a vomica.

Leube is authority for the statement that Rumpf has described a
of eliciting Wintrich's sign by which it can sometimes be demonstrated
fails if sought for in the ordinary manner. This consists in the perc
the chest over the supposed site of cavity during two successive inspir
ts, in such a way that one percusses during one inspiration whil
tient's mouth is closed and then repeats the stroke during the succe

inspiration when the patient's mouth is open and the tongue is outstretched. This is known as the *inspiratory change of pitch.*

Friedreich's Sign.—This is an elevation in the pitch of the percussion-note perceived during a deep inspiration. This like the foregoing is not always demonstrable even when a cavity exists, yet when conditions are favorable its detection enhances the value of other signs obtained by percussion and auscultation.

Cases are very infrequent as I know from personal trial which will furnish one or more of these percussion-signs with such distinctness as to establish the existence of a cavity, and therefore resort to auscultation is necessary. This means of examination may also prove uncertain. Yet, when the evidence obtained by auscultation is taken along with that of percussion, the weight of the two may be sufficient to turn the scales in favor of a vomica.

In the great majority of cases one hears over a deeply situated cavity nothing more definite than bronchial breathing and bronchophony. If this be sharply limited to a zone of tympanitic resonance and the voice be propagated to the ear with distinctness as if spoken directly beneath the bell piece of the stethoscope, it furnishes strong presumptive evidence of a cavity.

I have often detected a peculiarity of the vocal sound which when heard is generally held by me to be convincing, namely, a short whisper or sniffing sound which immediately follows the loud word and seems to be a whispered echo or resonance, only, unlike a real echo, it is not separated from the loud tone by an appreciable interval. This may correspond to what Leube terms "Nachklang" and considers very diagnostic. I believe this *after-breath,* if I may so call it, is never heard over a merely consolidated lung no matter how distinct may be the pectoriloquy under such conditions. At all events I have never so heard it and hence I regard this after-breath or whiff as proof of cavity.

Cavernous and amphoric breathing and voice are sufficiently characteristic and diagnostic under some circumstances provided they are corroborated by other signs. They may be present in other conditions and therefore are not pathognomonic.

Metallic tinkle is likewise of great value but may sometimes be heard in pneumothorax. This sound is a musical or clear metallic note something like the musical tone of a small tea bell and is produced by the falling of a drop of liquid from the wall of the large vomica into the fluid contents at the bottom. It may sometimes be detected after a hard coughing-fit.

Leube regards a metallic quality of the breath-sounds, of the voice, of the râles and of the percussion-note over a given area as excellent proof of the existence of a cavity.

If, in attempting to estimate the significance of any of the foregoing signs, the physician will only remember that tuberculous cavities are more frequent and more early in the upper than in the lower lobe, he will be greatly assisted in the diagnosis of this often very obscure condition.

The *differential diagnosis* of pulmonary cavities from bronchiectasis and pneumothorax will be found by a study of the respective chapters. There are circumstances in which the differentiation may be next to impossible. Yet if one takes into consideration the anamnesis, the symptoms and the results of painstaking and repeated chest-examinations a correct diagnosis is generally possible.

Prognosis.—In the great majority of instances individuals are seen at a time when hope of recovery is entertained, and consequently the first inquiry made of the physician after he has determined the nature of the case relates to the prospect of a cure. Therefore it will be well first to discuss the curability of pulmonary tuberculosis in general, and afterwards the individual conditions which affect prognosis.

Curability.—*Post mortem* records prove conclusively that tuberculosis of the lungs is capable of complete cure in the early stage and of a quiescence or relative arrest even in an advanced stage. It is probably well within the limits to say that from 40 to 60 per cent of all persons dying of accident, or from other than tuberculous disease of the lungs, exhibit *post mortem* evidence of healed lesions within these organs. These evidences consist in old cicatrices, calcareous nodules, zones of broncho-pneumonia that have undergone fibrosis and small cavities or caseous foci that have become encapsulated.

Such *post mortem* records are amply corroborated by clinical observation. Probably all physicians of wide experience can recall instances of persons who have recovered from pulmonary tuberculosis, the most of them after having been sent to some health-resort but some of them in their home climate. The majority of such survivors were in an early stage when they began to take their disease seriously in hand, but a few others showed signs of advanced disease. In almost every community, particularly in the West, can be found individuals who declare that they were given up to die of consumption forty years ago and are alive yet "in spite of the doctors."

In view of such facts and experiences, doubt concerning the curability of this disease cannot be entertained. The query now arises as to what percentage of cases can be expected to recover. Exact figures on this head are not easy to obtain, or at all events figures that apply to the great mass of such sufferers, since the statistics at command come mainly from sanatoria and have to do with persons placed in the most favorable environment for recovery.

It is the general consensus of opinion that pure and unmixed pulmonary tuberculosis is a comparatively benign affection, and hence it is the natural deduction that the *earlier the disease, the easier is its cure.* When, on the contrary, the disease has become moderately advanced, that is, when a pus-infection has become added to the tuberculous one, an arrest is still more difficult, and when the disease has gone on to softening and excavation there is very small prospect of arrest, even under the most favorable circumstances. Nevertheless, Dettweiler stated to me in 1893 that taking all his cases together without regard to stage of the disease he obtained an arrest in from 24 to 27 per cent.

The percentage of cures in early cases is undoubtedly very much higher, but figures based on the different stages of the complaint cannot be secured or are not reliable, for the reason that a strict division of cases into stages is oftentimes impossible. The latest statistics to which I have access are those derived from the report for 1903 of the *German Central Committee for the erection of sanatoria for consumptives, and published in the Geschäfts-Bericht of* 1904.

The results of treatment were considered from two standpoints, first the ability for self-support and second the recovery from disease. Moreover the 6,000 patients who were discharged and furnished the basis for the figures belonged to the working classes. They are, therefore, particularly valuable and instructive from all these points of view.

Of the 6,000 and over discharged 67.3 per cent were able to support themselves at their former employment; 7.1 per cent by some other kind of work; 14.6 per cent were able partially to support themselves; 11 per cent were not so capable of self-support. From the standpoint of recovery 87.7 per cent either recovered or were improved; 8.8 per cent were not improved; 3.1 per cent were worse; 0.5 per cent died.

The most favorable results were among patients whose disease was not advanced. Thus, whereas 87.7 per cent of those in all stages recovered or improved, it was found that of those in the first stage 95.2 per cent recovered or improved and of persons in the second stage 89.9 per cent showed the same results. It was also stated as noteworthy that a good many of the individuals who, when admitted, showed signs of advanced disease and subsequently were dismissed as not improved, nevertheless at a later examination were discovered to have meanwhile become capable of self-support.

Although the foregoing figures were published for the purpose of showing the immense benefit to consumptives of residence and treatment in public sanatoria, they yet illustrate the encouraging prognosis that may be held out to tuberculous patients if they avail themselves of the best means now known for their relief. Since, moreover, these figures were taken from the working classes of Germany who, it might be concluded, would not be likely to offer the best prospects of recovery, the conclusion would seem justifiable that among persons of abundant means to command everything needed for successful management, the percentage of recovery or improvement ought to be still greater.

The Relation of the Pulmonary Lesion to Curability.—Other things being equal the prospect of cure stands in direct ratio to the stage of the disease. In its incipiency, therefore, and in its early stage pulmonary tuberculosis offers a relatively good prognosis and individuals should be encouraged so to believe, *providing always they can and do avail themselves of all requisite measures.* It is essential to an intelligent forecast, therefore, that the physician be able to determine to what stage of the disease a given case may be assigned and hence the different stages will now be defined.

In the *incipient stage* belong cases that present no complications and mild general symptoms; namely, no temperature or not above 100.5° F., no expectoration, or if present scanty and mucous, a fair state of nutrition, and as regards the lungs, disseminated lesions in one lobe or in a half of two lobes shown by moderate dullness, diminished or broncho-vesicular respiration with or without fine bubbling or subcrepitant râles.

The element of time does not enter into the question of the stage of the disease, for a lesion still circumscribed to one apex and presenting few signs with mild constitutional disturbance may yet be in an early stage in spite of the lapse of many months. When the disease has spread to the second apex, even though the signs be circumscribed and the duration short, it is no longer incipient but may be in an early stage.

In the *second or moderately advanced stage* an arrest may still be possible although the prospect is not so good by several points as in the first one. The second stage is shown by the following:

The constitutional symptoms are less mild but still not severe. The temperature range is higher, to 101.5° F. it may be, expectoration is more abundant, muco-purulent, and nutrition has suffered appreciably but the patient is still not greatly reduced. Over the whole of one lobe or the greater part of two lobes are dullness, bronchial or broncho-vesicular breathing and copious small or medium-sized bubbling râles or clicks.

Not only are signs of disease in the lungs more extensive and pronounced, but the effect of the toxæmia is unmistakable. Time is not always an element to be considered in determining the existence of this stage, for a given case may have passed rapidly through the first into this.

In the *third or far advanced stage* the prognosis may be said to be unfavorable in the overwhelming majority of instances. Now and then a case may become arrested, but if so, it cannot have progressed far into this stage as shown by the state of general nutrition.

This stage is declared by more or less profound toxæmia, fever of an irregular often hectic type, purulent or nummular, usually abundant expectoration, and well-marked emaciation and feebleness. On the part of the lungs, dullness, circumscribed tympany; diminished, bronchial, cavernous or amphoric breathing; râles more or less widespread, musical dry or medium-sized and coarse bubbling; together with voice-sounds indicative of cavity, these various signs being distributed over more than two, often three, and possibly four lobes. Serious complications likely to be present.

It requires but brief consideration to convince one that such conditions are very adverse to recovery. Even if the lesions be not widely scattered, involving perhaps only two lobes, still the degree of accompanying constitutional disturbance is such as to preclude arrest of the pulmonary disease in nearly all cases. The different stages merge gradually into one another so that a strict classification is not always possible, but it may be said that the further the disease gets away from its incipiency, the less is the likelihood of an arrest.

Conditions of Curability Residing Outside the Lungs.—Some of these are found in the general symptoms and have been touched upon already, while others lie in individual characteristics, environment, complic. etc., as will be shown anon.

Emaciation is one of the most important of the constitutional eff regards prognosis. It may be held to be a rough but significant ir the intensity of the infection. Individuals are sometimes observe although in the incipiency or at most in the early stage of their dise yet greatly reduced in weight, presenting a degree of thinness ou proportion to the extent and intensity of pulmonary findings. In such the prognosis is not encouraging since they evince all too plainly the ravages of their infection. The same applies to wasting, even without fever, in the second and third stages, so that it often requires but a glance at the wasted form to convince the observer that the chances of recovery are few.

Fever is another symptom of potent influence on prognosis. *Cœteris paribus* the absence of pyrexia may be regarded as favorable. This remark must not be taken to apply to those cases sometimes encountered in which the temperature is subnormal, for such a low temperature may signify extremely low reaction and a grave degree of toxæmia. In general it may be held that the higher the fever the worse the prognosis, since it evinces an intense infection.

Likewise the outlook is bad when the fever has assumed the irregular type with wide variations in its extremes, which constitutes the so-called hectic fever. This type is generally seen in the late stage and marks the near approach of the end. It may, however, be symptomatic of some complication as a septic pyothorax, when the outlook is also bad. According to F. Wolff the prognosis is unfavorable when the acme of the fever-curve falls in the early afternoon and the remissions in the evening and early morning (Fraenkel).

On the other hand, the outlook is relatively most favorable when the temperature is normal in the morning and increases to only a moderate height in the late afternoon or evening. Such a temperature-curve may be said to come within the category of Strümpell's subfebrile form and not infrequently subsides gradually to the normal. Lastly, the so-called *febris inversus* affords a most unfavorable prognosis, since it is very apt to indicate an acute miliary tuberculosis.

The Condition of the Blood.—In accordance with the investigations and conclusions of Arneth, to which reference has been made in previous pages, one may obtain valuable and reliable prognostic data from a count of the leucocytes. If there is a persistent leucopæmia or if in the presence of a moderate hyperleucocytosis the increase is found to reside in the neutrophile cells belonging to the first and second groups, the ultimate prognosis is unfavorable, no matter how encouraging may appear to be the outlook as shown by gain in weight, and strength.

Digestive disorders likewise exert an unfavorable influence upon prognosis. The consumptive who has an unconquerable aversion to food cannot hope to recover, since he cannot successfully combat the tendency to wasting and exhaustion inherent in the toxæmia. Distress or an almost painful sense of distention after eating, complained of by some tuberculous patients, is also a bad indication, whereas the ability to eat heartily and digest large quantities of food increases the chances of recovery. *Diarrhœa*, whatever be its cause, is prognostically unfavorable since it tends to malnutrition and exhaustion.

Hæmoptysis is another symptom that may materially affect prognosis. It is believed by some that the hæmorrhagic form of pulmonary tuberculosis is relatively hopeful, since the recurring losses of blood diminish congestion. There may be some ground for such an opinion in the incipiency of the dis-case, but in confirmed phthisis and especially in the second and third st hemoptysis must always be looked upon as grave. The ulceration or rupt of a vessel of considerable size may at any time lead to fatal hemorrhag or repeated losses of blood may seriously deplete the strength or a septi broncho-pneumonia may be occasioned. I have known hemoptysis comple to reverse the previously encouraging outlook.

Erethism, by which is meant a great and uncontrollable excitability o the nervous and cardio-vascular systems, furnishes a prognosis that is a the least a dubious. Jaccoud holds this view, stating that whether th nervo-vascular excitability produces cardiac disturbances as palpitation only as irritability of the nervous system it is to be regarded as seriou diminishing the individual's prospect of recovery. This condition may so times be observed in the incipiency of tuberculosis and when present is alw viewed by me with much concern.

Fickleness or instability of temperament also exerts a detrimental influence over the prospect of recovery. A person of such type cannot be expecte to persevere for long in the monotonous routine and rigid measures laid dow for him by the phthisio-therapist, but before long will break through th restraint imposed and commit some indiscretion. *Steadfastness of purpos is essential in the successful fight against pulmonary tuberculosis.*

I once examined a young Jewess who, although plainly informed of th existence of tuberculous infiltration of one entire upper lobe, and of th mode of life requisite to its control, insisted that if she could not go t dances she did not care to get well. I have in mind another young woma who displayed total lack of serious determination to deny herself harmful social pleasures. Notwithstanding emphatic warnings this girl would go t dances and other social functions night after night, sit on the damp gras with her companions, go without rubbers and manifest other evidences o self-indulgence which kept her medical advisers in a perpetual state o apprehension.

The young man who persists in the use of cigars or cigarettes, who now and then indulges in a carouse or practices venereal excess or in various other ways kicks over the traces, so to speak, not only endangers but may actually ruin his chances of recovery. On the other hand, the consumptive who religiously obeys orders may turn what was a not very hopeful prognosis into a decidedly encouraging outlook.

Finally, in this connection it may be stated that a tuberculous subject who displays the peculiarities of his psychical nature described in the Analysis of Symptoms as evinced by injudicious acts of all kinds and emotional variability from despondency at one moment to unfounded hopefulness and gayety at another should never be given an unreservedly good prognosis, no m how mild his other symptoms, or how apparently encouraging his pulm lesion. He lacks the stability and fixedness of purpose necessary to th and persistent struggle ahead of him.

Home Environment.—By this term is meant all the conditions whether in home circle or business which make up the sum and subst one's daily existence. It goes without saying that an individual's of arresting his tuberculosis are to be gauged in large part by this standard. The fight against consumption is a very expensive one and, other things being favorable, a person's prospect of recovery depends upon his ability to command indispensable comforts and the best medical attention. *A person of limited finances is practically doomed from the start.*

If an individual is compelled to do housework or attend to his business when, because of fever and decreasing weight, rest in the open air is indicated, there is but small chance that the disease will be brought to a standstill. Likewise, what is to be expected from change of climate, if a person with confirmed tuberculosis of the lungs is obliged to seek some employment that may make considerable draughts on his strength or necessitate confinement within doors? It is plain that if a consumptive is thus handicapped the prognosis in his case is unpromising, to say the least.

Furthermore, if a tuberculous patient be compelled to remain in the miserable tenement house in which he lives or is otherwise forced to endure unhygienic surroundings, he cannot be expected successfully to resist the incursions of his archenemy, no matter how early this latter be discovered or however trifling the constitutional symptoms may appear to be. In a word, *to be successful, the fight against pulmonary tuberculosis must be waged intelligently, determinedly and under the most favorable conditions,* and the absence of such conditions renders the prognosis hopeless.

Complications.—The influence of the various possible complications upon the course of this pulmonary affection has been already considered, and hence but few additional words are needed here. Pneumothorax, with or without the supervention of serum or pus, affects prognosis most unfavorably and for the most part brings about the patient's death more speedily than otherwise it would have come. Exceptionally, however, this complication may be recovered from and the phthisis be brought to a partial arrest or rendered less rapid in its progress.

A pleuritic effusion may or may not prove a very serious matter depending on its amount and the strength of the patient. It may favorably affect the primary lung disease through compression and immobility of the lung, but such is not to be counted upon in considering prognosis. Repeated pleurisies may be held to indicate an unfavorable outlook as showing no tendency of the primary affection to quiescence.

Involvement of the larynx, intestines or other organs renders prognosis doubtful or absolutely hopeless according as the secondary localization is amenable to successful treatment or otherwise. In the majority of cases these complications render the prognosis unfavorable to recovery. The person with pulmonary tuberculosis in any stage has enough to contend with already and the addition of any other burden lessens by just so much his prospect of regaining his health.

Probable Duration.—To venture to set a time limit to a case of chronic pulmonary tuberculosis has always seemed to me most unwise. It is not at all infrequent to meet a consumptive who states that the eminent Dr. —— gave him but six months or a year to live and yet here he is in better health than when the unwise prediction was made. The reason for our inability to state the duration of any given case lies in the fact that we cannot always estimate the power of a person's resistance or foresee how rapidly or how slowly the local changes may advance. We may state approximately the probable duration from data furnished by a large number of cases, but even in this regard reliable figures are difficult to obtain.

The disease does not run a definite course as do most acute affections and in its progress is so influenced by outside conditions that very great differences are found as regards duration. Now and then instances are met with in which the disease has persisted for ten, fifteen, twenty or even thirty, yes, forty years. They are spoken of as "cases of old-fashioned lingering consumption." As most of such cases I have heard of existed prior to Koch's immortal discovery I have been inclined to look upon some of them, at least, as not having been genuine tuberculosis, but cirrhosis or a chronic catarrh of the pharynx and trachea, or large bronchi, etc. Nevertheless, that some cases of true consumption may last for many years cannot be denied.

The duration is determined by many factors and hence the figures on which estimates are based do not always apply to all classes of individuals. Thus, Dettweiler gave seven years as the average duration in his cases, but, as remarked by Cornet, his observations were made among consumptives of the better class socially and hence are probably too high. Cornet from a study of approximately 800 cases of the poorer class obtained an average duration of two to three years. Reiche from a study of the disease among working people who were not treated in sanatoria gave forty-three months as the average duration from the beginning of the complaint (Fraenkel).

Other figures and opinion might be quoted, but it seems scarcely worth while since they only emphasize the great uncertainty and variability of the duration as governed by circumstances in each case. It may be added, as a matter of interest merely, that according to Cornet the period of latency, i. e., from exposure to infection to the outbreak of symptoms, may vary from six months to one year on the average, although it is possible for even years to elapse.

CHAPTER XXVIII

ACUTE PULMONARY TUBERCULOSIS

As remarked by Cornet, it is not always easy to classify all cases of pulmonary tuberculosis as regards acuteness or chronicity. In some the course is so rapid and death results in so short a time that they fall plainly into the category of acute. In other cases the disease pursues so protracted a course, extending over years, that the term of chronic phthisis is most applicable. In others again the course of the complaint is embraced within a number of months, five, nine or twelve, and one is in doubt just where to place them. They are on the border line, as it were, and so may not inappropriately be called subacute. In general it may be held that acute cases terminate inside of six months.

Two main divisions may be made of acute cases, (1) the acute pneumonic form and (2) the acute miliary form of pulmonary tuberculosis which is, as a rule, a part of a general miliary process. Again the pneumonic form is divisible into two groups, (*a*) the lobar form and (*b*) the lobular or broncho-pneumonic form. Of these the latter is the more common and yet neither occurs anywhere so frequently as does the chronic type of the disease, their frequency being variously estimated at from 2.6 to 19 per cent of all cases of pulmonary tuberculosis (Fowler).

(1) ACUTE PNEUMONIC TUBERCULOSIS

Morbid Anatomy.—In this form of acute tuberculosis of the lungs the inflammation leads to an exudate rich in cells which instead of showing a tendency to resolution undergoes caseation. Hence it is sometimes designated acute caseous or acute scrofulous pneumonia. Tubercles are not always recognizable and hence arose the dual theory of pulmonary consumption held by Virchow and adherents of his school. The infiltration is uniform and gelatinous, at first having a gray or grayish white appearance, but soon becoming yellowish and cheesy.

If it involve an entire lobe (Fig. 44) it may, when examined early, so closely resemble a primary fibrinous pneumonia as to be differentiated with difficulty. Even in the disseminated or broncho-pneumonic form the areas may to the naked eye look like patches of ordinary broncho-pneumonia (Fig. 49). In both forms, however, by aid of the microscope it is usually possible to recognize the cellular infiltration of bronchial walls of the alveoli and of the connective tissue separating the various structures, in other words, to recognize a uniform infiltration with tubercle.

In cases of disseminated small patches of broncho-pneumonia careful study may disclose tiny miliary tubercles surrounded by zones of inflammatory exudate separated by deeply injected parenchyma, so that to the unaided sight the appearance is not unlike that of red hepatization that here and there is passing into the gray. The overlying pleura is hyperæmic and inflamed or covered by a thin exudate and it may show miliary granules.

The character which particularly distinguishes this pneumonic form of tuberculosis is its tendency to rapid caseation and excavation, and yet in some instances there is an attempt at formation of fibrous tissue without softening and destruction. These are the cases that run a longer course and pass into the subacute or chronic form of caseous pneumonia. In all there is very slight or no attempt at repair by absorption of the inflammatory exudate and recovery by resolution.

Ætiology.—The disease may occur at any age but is most frequent among young adults and in children. It may be primary, or at least appear to be such, but is for the most part secondary to some antecedent lesion at the apex, as an old cavity, or to a caseous bronchial gland which has softened and discharged into a bronchus. It is, therefore, in most cases an example of aspiration pneumonia. Such was the origin of a striking and typical instance seen in a Negro girl at Cook County Hospital in the Winter of 1905.

Symptoms.—This form of pulmonary tuberculosis may set in abruptly with rigor and other initial features of a primary pneumococcus pneumonia or its development may be insidious and gradual, having been preceded by slight cough and *malaise*. In the former event particularly, the clinical features are so like those of a frank croupous pneumonia that at first the physician fancies he has to do with that disease.

Chill and pain in the side are soon followed by rise of temperature to several degrees above normal, by cough, expectoration, perversion of the pulse-respiration ratio, perspirations and prostration, and the physician finds the usual signs of pneumonia of an apex or rarely of a lower lobe.

Chill may be single and pronounced or there may be a repetition of chilly sensations. In some cases no definite history of rigor can be elicited.

Pain in the side may or may not be complained of, but often there is a sharp stitchlike pain that announces the onset of the disease.

Fever is a marked feature and may be of a high continuous type for the first week or so. In other cases it is remittent or even irregular, and as the disease advances this type of the pyrexia becomes more pronounced and it is unmistakably of a hectic character.

Cough is not more frequent at first or more severe than in other forms of pneumonia. As softening occurs, however, and the bronchial mucosa becomes irritated and inflamed by the bacilli-laden sputa, cough grows worse and
greatly disturb the patient. If laryngitis ultimately complicate the case
is not very unfrequent, this condition proves a prominent feature.

Expectoration is of a mucoid or rusty appearance the same as in any pn
monia and indeed may be indistinguishable at first from that of crou
pneumonia save by microscopic examination. Occasionally there may be
hæmoptysis as an initial symptom. As the disease progresses the sputa as

a yellowish puriform color and in some instances, as first pointed out by Traube, may have a distinctly green hue.

With the advent of softening and excavation bacilli appear often in huge numbers and there are elastic fibers or fragments of pulmonary tissue. At this time again hæmorrhage is an occasional event being due to the ulceration of a blood-vessel attending rapid lung destruction. Such hæmoptyses are often large and greatly to be dreaded.

The *pulse* is usually rapid and yet early in the course of the affection is not relatively so accelerated as are the respirations, so that there is a perversion of the pulse-respiration ratio the same as in true fibrinous pneumonia. At a later stage, however, the pulse grows more frequent and of low tension, 120 to 130, and in consequence of exhaustion remains rapid even during periods of remission in the fever.

Dyspnœa, in the sense of a subjective want of sufficient air, does not, according to Fraenkel, appear so prominently in this as in the pneumococcus form of pneumonia, and in this utterance I must concur. Indeed I can recall at least two instances of acute tuberculous inflammation of the apex in which, notwithstanding the involvement of the entire upper lobe, the patients made no complaint of dyspnœa. In fact their breathing, although accelerated, did not impress me so much as did the frequency of the pulse.

Prostration and rapid emaciation mark the more acute cases, but delirium is not usual. Anorexia and thirst are present as in other febrile affections, the bowels may or may not be confined, and the urine is scanty and contains urates but no albumin. It may show the diazo reaction.

Physical Signs.—Examination of the lungs discovers dullness, most commonly of an upper lobe, and usually more pronounced and to a lower point behind than in front. The breath-sounds are bronchial and in the commencement of the process accompanied by crepitant râles, undistinguishable from those of acute fibrinous pneumonia. Dullness and bronchial breathing are more intense in the lobar or confluent form of this type and yet disseminated areas of broncho-pneumonia may be so thickly set together as to produce these signs in an outspoken manner.

As has been shown, the disease in question resembles an ordinary lobar pneumonia so closely that the physician at first believes he has to do with a case of that kind. Accordingly, as the seventh day approaches he begins to look for crisis. This fails to occur, however, and examination of the lung detects here and there subcrepitant râles, which the physician hopes may indicate beginning resolution. Such does not take place however and the râles grow daily more numerous and of a larger and more sharply clicking character.

Dullness grows a trifle less intense because of the development of a tympanitic quality especially below the clavicle, whereas behind, the note may remain as dull and the sense of resistance as great as before. It is about this time that the sputa begin to show bacilli and elastic tissue and to become much increased in amount.

There is now no difficulty in diagnosis. Even without a microscopic inspection of the sputum the nature of the case becomes apparent from the protracted course, the increasing exhaustion, the change in the physical signs which, together with the profuse purulent sputum, denote softening of the pneumonic area. At length, over a greater or smaller area in the infraclavicular region appear unmistakable signs of a cavity, a circumscribed zone of high-pitched tympanitic note that may show Wintrich's or Gerhardt's change of pitch, tubular, cavernous or amphoric breathing and corresponding voice-sounds with possibly metallic sounding râles.

Signs like the above usually develop slowly and take several weeks to become fully established, but now and then a case is seen in which a portion of the pneumonic lung breaks down abruptly and *en masse* forming an abscess. In such an event the pus and *débris* may be suddenly discharged *per os* when the signs of vomica speedily appear.

The *duration* of the disease varies within wide limits. In some cases a fatal issue occurs in two or three weeks from its commencement. These are what are termed Galloping Consumption. Time is not given for extensive softening and signs of cavity do not appear. In other cases the term Phthisis Florida may be still appropriate but the course of the disease is less rapid and may cover several months.

The *termination* of the most acute cases is in death from exhaustion or pulmonary hæmorrhage. In the subacute cases the lung originally involved may, after undergoing rapid excavation, show signs of arrest of the destructive process, and the improvement in the general as well as in the local condition may arouse hope that the disease may be arrested or at least pass into a chronic state.

In the Summer of 1897 I had charge of an instance of this kind. A young married lady developed a tuberculous pneumonia of the left apex which at first ran a very stormy course. After the development of cavity the disease appeared to lessen in violence, râles grew fewer, expectoration diminished, fever abated and strength was gained. Three or more months had now elapsed, and hope was entertained that the disease would become chronic and so afford a chance of ultimate recovery.

Suddenly the patient complained of pain on the opposite side, fever increased, cough became more urgent and examination discovered dullness and bronchial breathing over the other apex. This pneumonia pursued the same rapid course as did the former, the lung broke down rapidly, what little strength had been gained was soon lost and death took place in about nine months from the onset of the first attack. This case developed during lactation and about two months following child-birth.

An instance of the more chronic kind is the following: A young married man of twenty-nine with a tuberculous family history developed signs of pulmonary disease early in the Summer of 1894 and was sent to Colorado by the physician whom he consulted. After having been there for a month o his condition grew so rapidly worse that he was sent home and the phys whose advice he had first sought gave him but a month to live.

I happened to be at the station when he was helped from the cars friends. His weakness and emaciation and general look of the consu certainly appeared to justify the prognosis that had been given. Tha day I was asked to see him and take charge of his case.

The left lung was dull throughout with exception of a small area of high-pitched tympany in the middle of the infraclavicular region over which was distinct bronchial breathing. Elsewhere the breath-sounds were either indeterminate or feebly broncho-vesicular while râles were scattered and not numerous. The pulse was frequent and feeble, respirations were increased but not dyspnœic, fever was remittent, appetite was gone and prostration was marked. It certainly was an unpromising case.

The young man was kept at absolute rest out of doors and forced feeding was instituted. Very little medicine was prescribed, being only such as was indicated by special symptoms. The results were surprising. Treatment was begun the fore part of August and by the middle of October his condition had so greatly improved that he was permitted a small amount of exercise in the house.

He had taken on a little flesh but had not regained his full strength, and continued to cough and expectorate although much less than some weeks previously. About this time he developed a complication which for a while retarded progress. This was a thrombo-phlebitis of the left femoral vein that led to considerable œdema of the foot and leg. At length, with rest and elevation of the extremity this symptom disappeared and the patient became able to walk about a little.

Meanwhile the lung-findings had not materially altered. The left lung still showed dullness throughout with exception of the tympanitic area in the infraclavicular region. The breath-sounds were variable, bronchial in some areas and indefinite or broncho-vesicular in others, while râles were very few and clustered mainly about the vomica.

By the middle of November his condition had so greatly changed for the better that it was decided to send him to Asheville, N. C., in the hope that a Winter passed in a mild climate permitting life out of doors might aid powerfully in bringing the disease to an arrest, as it was thought that the diseased lung was tending to fibrosis. At first the change appeared to benefit the invalid considerably, but after a while he seemed to have reached a standstill. Thus was passed the Winter and Spring came on. The patient now showed signs of failure and at length, an obstinate diarrhœa having set in, he died something like a year after the onset of his malady.

Diagnosis.—There is no difficulty in recognizing the existence of the pneumonia, but the determination of its nature in the commencement is almost impossible. Its resemblance to the fibrinous variety is so close, particularly when an entire lobe, e. g., the lower, is involved, that even the most experienced physician is likely to mistake it for an instance of that sort. There may be some fact in the previous history, as of chronic cough or of a period of failing health or exposure to infection that may arouse suspicion of its tuberculous nature.

Nevertheless, a tuberculous individual may develop a croupous pneumonia and this consideration serves but to increase the perplexity. The pulse may be relatively more frequent and there may be less dyspnœa than would be expected in a case of pneumococcus origin, but it is not until the physician detects the signs of softening and notes the failure of the case to exhibit evidence of resolution and convalescence after the time when such should occur in true fibrinous pneumonia, that he obtains data pointing with certainty to the tuberculous nature of the process.

In this matter of differentiation from acute fibrinous pneumonia it may be stated that if the leucocytes do not show a great increase over the normal, the process is probably tuberculous; and should the neutrophile cells of the first and second groups (Arneth) be disproportionately increased the pneumonia may be pronounced probably caseous, for although a pneumococcus pneumonia may show similar blood findings, such are extremely rare.

The discovery in the expectoration of bacilli and shreds of pulmonary tissue is, of course, the one sure means of diagnosis, but unfortunately is not available in the early days of the disease. Only exceptionally can the process be differentiated from acute croupous pneumonia before these germs make their appearance in the sputum; and prior to the discovery of the large and fine clicks that indicate softening the diagnosis of a tuberculous inflammation of the lungs can only at the best be inferential.

Prognosis.—In the majority of cases this is hopeless, all efforts to stay the progress of the disease being fruitless. In less acute cases there is hope that with the breaking down and casting out of the diseased focus the malady may become subacute or even chronic and then, with proper management, allow the lung to heal. Such a fortunate outcome is rare, however. Rapid destruction of the lung carries with it the possibility of erosion of a good-sized vessel and alarming if not fatal hæmorrhage. The occurrence of complications, as tuberculosis of the larynx or intestines, renders the prognosis still more unfavorable.

(2) ACUTE MILIARY TUBERCULOSIS OF THE LUNGS

This forms but a part of a general tuberculosis and is often concealed by the symptoms referable to that condition. Occasionally, however, cases are encountered in which the pulmonary manifestations predominate and one is justified in the designation of acute miliary tuberculosis of the lungs. For this reason it is considered practical to recognize this clinical variety of the disease.

Ætiology.—General miliary tuberculosis results from the inundation of the system by a vast number of bacilli that have been discharged into the blood-stream from some caseous focus situated in the wall of some vessel, e. g., the thoracic duct, or in immediate proximity thereto. The germs lodge in all parts of the body and grow and multiply. The clinical picture that ensues is, therefore, determined by the structures that bear the brunt of the attack, as it were.

Morbid Anatomy.—In some cases the lungs appear studded thro
with minute gray nodules without there being any evidence of previous l
In such instances there is apt to be an acute pulmonary emphysema
is shown by increased volume of the anterior borders which during li
rise to exaggerated resonance. In other cases there are old caseous
one or both apices and in such the general and pulmonary miliary dis

tion forms a terminal event. This is one of the possibilities that should always be kept in mind in any case of consumption. It is not easy or possible in every instance to discover the focus which was responsible for the inundation of the system with bacilli.

Symptoms.—As already said, the clinical picture depends upon the extent and predominating seat of the miliary granulations. It does not belong to the province of this work to describe the various clinical manifestations that result from general tuberculosis and yet it may not be amiss to remind the reader that aside from the pulmonary form there are two other chief clinical varieties of general miliary tuberculosis, namely, the typhoid and the meningeal.

In the **typhoid form** there is an intense general infection characterized by profound prostration, high and irregular or inverse type of fever, rapid feeble pulse, enlargement of the spleen and often of the liver, petechiæ closely resembling rose-spots, indefinite catarrhal manifestations on the part of the lungs and toward the end a tendency to delirium or coma. In fine, the picture is so like that of enteric fever that only by means of a blood-examination can the differential diagnosis be made with certainty.

In the **meningeal form,** seen most often in children, the symptoms are those of a meningitis and are not likely to be mistaken. The difficulty lies in always recognizing the nature of the meningitis. The condition arises less suddenly than is the case with meningitis of other origin, and careful inquiry into the history will usually enable one to arrive at a correct understanding of the case. There is also in children the hydrocephalic cry, and ophthalmoscopic inspection may recognize miliary tubercles in the choroid.

In the **pulmonary form,** as is implied by the name, the symptoms on the part of the lungs outbalance those of the general infection. The two most characteristic features are *dyspnœa* and cyanosis. The former may be shown by rapidity of breathing rather than by actual air-hunger, and yet this latter may be extreme. Thus I recall the case of a young man who was admitted to my service in Ward 4 of Cook County Hospital in the Winter of 1891 and 1892.

Details of the case are not now at hand, but I recall distinctly his great rapidity of respiration and his sense of air-hunger which compelled him to sit up in bed and often on its edge. The lungs were hyperresonant and he displayed very little cough. The autopsy showed the lungs absolutely stuffed with miliary and submiliary nodules from apex to base.

Cyanosis may in some cases be well marked while in others it is apparent only by slight blueness of the lips, cheeks and tips of the fingers. As this cyanosis depends upon defective aeration of the blood, it may be present in cases of extensive caseous infiltration without miliary granulations.

Fever is also a symptom of most cases but displays no especial type. It is likely to be irregular and to exhibit wide fluctuations. It may show the inverse type as in the case of a young woman seen in Cook County Hospital who at the same time showed considerable respiratory embarrassment.

Fraenkel states that cases of miliary tuberculosis of the lungs are now and then seen which display very slight or no febrile temperature and that Lichtenstern has observed such instances chiefly in elderly persons. In such there may be cardiac incompetence and œdema.

Cough may vary considerably in frequency and severity depending largely on the existence or not of antecedent changes and the intensity of the symptomatic bronchitis. In the majority of cases cough is not a very conspicuous feature.

The *expectoration* in cases of pure miliary dissemination in the lungs, that is, without old caseous foci, is not copious and is catarrhal or mucoid. In the strictly miliary cases it is not likely to contain bacilli owing to the fact that the nodules do not caseate and discharge into a bronchus. If the attendant hyperæmia is great the sputum may be blood-tinged or rusty (Fraenkel).

The *pulse* is likely to be rapid and of low tension, and *prostration* becomes a marked feature, particularly toward the close of the illness.

Physical Signs.—In cases in which the tuberculosis shows itself only by miliary nodules the examination of the chest is likely to furnish very negative results. Pulmonary resonance may be retained throughout or may even be exaggerated owing to acute emphysema. In other cases the active congestion attending the eruption of tubercles may occasion slight or indefinite impairment in scattered areas, particularly toward the bases.

The breath-sounds may remain vesicular or seem rougher than normal, while here and there may be heard a few catarrhal râles which, according to Fraenkel, may change in kind and place from time to time. In a suspected case such slight changes are of great significance.

In other cases there may be more or less pronounced dullness at an apex with the changes in the respiratory murmur and the adventitious sounds indicative of caseation or excavation. While in the remaining portions of the lung the findings may be as indefinite as those already described.

Diagnosis.—In many cases this must be inferential, but is rendered probable by the conjunction of dyspnœa, cyanosis and irregular, perhaps inverse, type of fever together with cough and catarrhal expectoration. Should such serious symptoms arise in an individual showing dullness and bronchial breathing at an apex the likelihood of a miliary dissemination in the lungs becomes very great.

The disease must be differentiated from typhoid fever and from influenza. We have a reliable means of differentiation from the former in the Widal reaction, but, this failing, careful attention must be paid to the respiratory organs. In tuberculosis the breathing is likely to be more frequent and the cyanosis greater. An emphysematous note anteriorly with indefinite ar of impaired resonance at the bases is in favor of tuberculosis as agai typhoid.

In influenza there may be a history of the disease in other member the family or in epidemic form in the community. There may also be dence of more or less catarrhal inflammation of the upper air-passages. spleen is not apt to be enlarged as is likely to be the case in miliary t

culosis and the diazo reaction is not present as is the case in the for
tion. Microscopic examination of the sputa may reveal influenza g
remarked by Fraenkel, the chief perplexity arises when the *grippe*
person who is already tuberculous.

Prognosis.—This is hopeless. We possess no means of arrestin
tuberculosis and hence a fatal termination of the case is only a q
time, in most cases of only a few weeks.

CHAPTER XXIX

PREVENTION OF PULMONARY TUBERCULOSIS

In Regard to the Public.—Nearly a quarter of a century has passed since the recognition of the tubercle bacillus as the specific cause of tuberculosis which, in its pulmonary form, is the "Great White Plague" that has destroyed untold millions of the flower and pride of nations. The hope at first aroused, that some therapeutic measure would be discovered capable of antagonizing the germ in the animal organism and of thus curing consumption, has now been practically abandoned.

Laboratory workers are interested in methods of establishing immunity and in questions pertaining to the intercommunicability of human and bovine tuberculosis. The profession at large and health boards in particular are concerned with the best means of restricting the spread of the infection. The laity is becoming aroused to the danger lurking on every hand, laws are being enacted, societies are being organized for the study and prevention of this widespread evil, in short the watchword has become Prevention and not Cure.

Meanwhile the condition of those already smitten by the plague is not neglected. Private and public sanatoria and hospitals are springing up in all parts of the civilized world, so that, to the optimistic, the time seems not very far distant when the various agencies now under way will control, if not eradicate, this terrible scourge in the same way as has been done with the bubonic plague, cholera and other epidemic maladies. To the accomplishment of this great end, however, it is necessary that physicians collectively and individually coöperate with sanitarians and other public-spirited citizens in attempts to educate the people and influence legislators to the adoption of all measures considered necessary for the attainment of this mighty object.

Unfortunately complete harmony of view does not yet prevail on many points and it is openly charged that in the indifference or ignorance or both, on the part of the medical profession, lies one of the greatest obstacles to prevention of this "Pestilence that walketh in darkness" and this "destruction that wasteth at noonday." It is thought important that in this chapter this burning question of the day be discussed in all its aspects, and ther
the various measures for prophylaxis will be considered as they affect
the community and the individual.

The Education of the Medical Profession.—It may seem presumpti
some of my readers for me to declare that if pulmonary tuberculosis i
to be stamped out it must be through a more intelligent appreciati
the part of physicians, of its infectiousness and communicability ar

is more, of its entire preventability. In his address on Administrative Control before the Phipps Institute in Philadelphia Dr. Herman M. Biggs stated that Robert Koch went further than this by declaring that the disease would not cease to exist until the present generation of medical men had died out.

They must be replaced by physicians thoroughly imbued with the bacteriology of the tuberculosis problem and therefore with its communicability and preventability. They must believe that it is propagated through contact and not inheritance. Dominated by this conception they will then become disseminators of this doctrine among their patrons. They will no longer leave the work of preventing this terrible plague to health boards and to the enlightened of the laity.

The Education of the People at Large.—This is fundamental but must of a necessity come slowly. There are some individuals who already appreciate the truths underlying prevention but the great mass of the people is still densely ignorant. Unfortunately there are many otherwise intelligent persons who do not seem to realize the full significance of the fact that consumption is a germ disease. Knowledge on the points concerned must be disseminated by the distribution of leaflets, articles in the newspapers and magazines, and by conspicuous placards posted up wherever they may attract attention. Teachers in particular should be instructed and then required to teach the truths to their pupils. In these and kindred other ways to be enumerated the attempt must be made to reach all classes of society. Physicians everywhere should coöperate by pointing out plainly and simply the following facts:

(1) Tuberculosis is caused by a specific germ and is communicable therefore, under certain conditions, from man to man and from animals to man. These conditions are (*a*) that there be a discharging or ulcerating lesion, (*b*) that the discharges from such sores contain the specific germ, the tubercle bacillus, (*c*) that the bacillus shall gain access to the animal body.

(2) Pulmonary tuberculosis becomes an ulcerating lesion when there is breaking down of the lung and bacilli are being discharged by the expectoration. Under these conditions, therefore, consumption is communicable.

(3) Tuberculous sputum is a menace whenever it is not effectually destroyed but is permitted to fall on floors, garments, etc., to soil the lips, face, beard or hands, or in any other way to mingle with the dust or atmosphere so as to be taken into the mouth or lungs of persons who attend the invalid or subsequently inhabit the room or building.

(4) Tubercle bacilli are capable of retaining their virulence for months and years under suitable conditions of dirt, darkness and moisture, but are readily destroyed by exposure to sunlight, ventilation and cleanliness.

(5) Consumptives are a source of danger when their expectoration is not destroyed and conversely are not a source of danger when they exercise certain precautions as regards their sputum.

(6) There is no need whatever of alarm on the part of the public so long as precautions are taken; and public sanatoria or hospitals for the cure of consumptives are not a menace to the community, in fact are not to be

considered for an instant in comparison with the risk to the public of allowing consumptives to roam about expectorating when and where they will.

Popular Lectures.—It would seem an easy matter to instruct the public in the truths just stated, but in reality it is not. The physician can and does inform an occasional invalid or family of the infectiousness of pulmonary tuberculosis and of methods for its prevention, but the great bulk of the people is left to acquire such knowledge by chance. Such should not be the case; and as the medical profession is the guardian of the public health and is aware of the infectious and communicable nature of this universal scourge, medical men everywhere should take active steps in the direction indicated.

In most of our large centers of population popular lectures are given every Winter in which are presented the various facts so necessary for the people to know. These lectures are given under the auspices of some society and are generally delivered by physicians. To the shame of the medical profession, however, it must be stated that in many instances these lecture courses have been arranged by public-minded laymen, and persons non-medical have displayed more interest in them than have the doctors of the community.

Such should not be. Physicians everywhere, in hamlets and rural districts as well as in the cities, should participate actively in this crusade against tuberculosis. For the most part the people are eager for information, for there are very few who have not suffered from this terrible plague. These public addresses should present facts and figures showing the immense cost to the state and community of consumption, as well as its methods of spread and prevention so that a public sentiment may be created which will influence legislators to enact laws and make appropriations for the suppression of this disease.

Regarding the economic loss entailed by pulmonary tuberculosis let me quote from Dr. Herman M. Biggs who has for many years been actively concerned in the sanitary problems confronting New York City. "It may be conservatively estimated that each human life at the average age at which the tubercular deaths occur, is worth to the municipality $1,500. The cost of each life at this age is usually more than this. This gives a total value to the lives lost annually of $15,000,000." This is based on a death rate of 10,000 in New York City annually. "We may further assume that for an average period of at least nine months these persons are unable to work and must be cared for. The loss of their service during this period may be estimated at $1 a day, and the cost of food, nursing, medicines, attendants, etc., at $1.50 more per day, making a further loss of $2.50 per day, for each person dying, for a period of 270 days. This gives us a further loss to the municipality of $8,000,000, making a total annual loss to the city from tubercular diseases of at least $23,000,000. It has been estimated that in the United States annually not less than 150,000 deaths are caused by the tubercular diseases, and, estimating the value of these on the basis just given, we have an annual loss to the country of more than $330,000,000."

It is believed by Dr. Biggs that if but a small part of this amount of money were obtainable for the purpose of combating this disease, it would result in the practical eradication of the scourge, his conclusions being based on the almost unexampled reduction in the death rate from tuberculosis that has been achieved in New York with a very meager expenditure of money. Is it not worth while, therefore, that the people and their legislators be educated in the prevalence and prevention of this costly disease?

The Formation of Antituberculosis Societies.—The education of the profession and of the people must be made efficacious by the formation and affiliation throughout the country, and indeed throughout the world, of associations for the study and prevention of tuberculosis. This has been done already to a limited extent, but the interest in and the membership of these various national, state and municipal societies are as yet wholly inadequate to the needs of the movement. I would urge every reader of these pages, therefore, that he become actively interested in the work of those already established and in the formation of others where such do not exist. The membership of such organizations should not be limited to the profession, but should include as many of the laity as can be induced to join. The objects should be to instruct, to raise funds, to obtain needed legislation and to enforce laws looking to the prevention of this malady.

There will always be some individuals who receive the word gladly and endeavor to carry it out faithfully. For such, nothing more is necessary than instruction, but there are many others who refuse to carry out the dictates of humanity or to obey the mandates of the law. They resent what they consider interference from others and, in the matter of expectoration, e. g., display an utter disregard for the wishes of others and seem to glory in arousing a feeling of disgust in the decent-minded who chance to be their neighbors for the time being.

Antispitting Laws.—One of the objects of law is the protection of society and the individual against injury by the vicious or self-seeking, and, without law properly enforced, communities would soon lapse into barbarism and anarchy. The individual must subordinate his wishes and his notions of personal liberty to those of the majority. He cannot be allowed to commit acts that endanger public safety. Society has recognized this in regard to certain infectious diseases, as small-pox. The right of the authorities to isolate the small-pox patient for the sake of protecting the community is only questioned by a few cranks who likewise oppose and resent vaccination.

Influenced by the agitation of the question of the prevention of pulmonary tuberculosis, health authorities have quite generally secured the passage of laws looking to the stoppage of expectoration in public places where it can prove dangerous to the health of the people. There should be not only no objection to such legislation by right-minded citizens, but an active coöperation with the authorities in the enforcement of such wise provisions of the law. Whenever they detect an ignorant or unwilling person violating the ordinance they should enter a complaint and aid in his arrest and punishment.

At a recent meeting of The American Climatological Association one of the members related the instance of a consumptive who, having been permitted to leave the institution of which he was an inmate on Election day, had become intoxicated and in returning in a street car was seen by the physician to expectorate on the floor. Now here was a man who, through ignorance or carelessness reënforced by intoxication, was sowing broadcast seeds of disease. Can there be any doubt of the wisdom and necessity of laws for the control of such dangerous persons?

Educate the people regarding the cause of pulmonary tuberculosis and the ways in which it is spread through the community and they will hail gladly any and all measures calculated to protect them and theirs from the liability to infection. Let them once come to realize that a consumptive who spits in public places is to be placed in the same category as the small-pox patient running at large, and they will see that appropriate laws are passed and enforced not only in our large cities but in small towns and hamlets.

Administrative Control.—It is still a vexed question, how far the state or community should be allowed to control the actions of individuals and corporations in this matter of prevention of pulmonary tuberculosis. It would seem as if the authorities should have the right to restrain the liberty of such dangerous consumptives as the one cited above by confining them within suitable institutions provided for their care. Yet there is still a strong public sentiment, shared by many of the profession, that it is unnecessary and cruel to adopt measures which would practically brand the tuberculous as an outcast and reduce him to the position of the leper of olden times.

There are others who believe that the enactment of rigid laws will create an altogether unnecessary fear in the minds of the people regarding the contagiousness of the disease. They point to the hostility aroused in certain localities to the erection of sanatoria and hospitals for consumptives lest they prove a source of danger to themselves and their families. To this it should be replied, that such unfounded hostility emphasizes the necessity for public education and is no argument at all against the wisdom of proper administrative control.

In answer to the objection that the consumptive is likely to be shunned as was the leper of old it may be said, as long as the tuberculous will obey the law against spitting and will exercise the precautions to be laid down by the physician, and subsequently to be described, he is not a source of special danger to his fellows, and the properly educated public will not so regard him. When, however, he ignorantly or willfully disregards the safety of the community, then he should be looked upon as is the leper and should be treated as an outcast.

The interest of the private individual is of far less importance than is the good of the public. If the consumptive is made to see that only by obedience to law can he escape the fate of the leper, then he will comply with health ordinances and cease to be an object of distrust and police surveillance.

Health boards should have full powers vested in them by law to co corporations to provide cuspidors for their patrons and employees an have their premises and public conveyances thoroughly cleaned and d fected. This applies particularly to street-car and railroad companies.

There is a certain wholesale house in Chicago which enjoys the rep tion of being especially kind and generous to its employees. As it happ I have been called on to examine several of the clerks from this house the past year and in every instance I have found the individual to be suffering from advanced tuberculosis. Upon inquiry as to the disposal of their sputa I was horrified to learn that, in one instance at least, it was the habit of the clerk to expectorate upon the floor and that his fellows did the same. No cuspidors were provided by the firm. I then wrote to the head of the house stating the facts in the case and the dangers to himself and every other habitué of the premises, and suggested certain measures to be adopted. The result was that my suggestions were thankfully received and adopted.

In many cases it is ignorance or thoughtlessness that is responsible for neglect of proper precautions, but in others it is greed that leads corporations to disregard public health; and whenever such is the case, the lawful authorities should step in and compel them to do otherwise. There are many other evils besides public spitting which should be corrected by law, and the most serious of them will now be considered.

Factory Inspection and Control.—It was shown in considering ætiology how serious an element in the maintenance and spread of this infection are the conditions prevailing in certain factories such as sweatshops. Public welfare as well as the health of the garment workers demands that these abuses be corrected by the power of law as vested in boards of health. The Cigarmakers' Unions have reduced the death rate of their members and added to their life-expectancy by improving the sanitary condition of the factories, by shorter hours of work, etc. There is no reason why similar beneficent results should not be obtained in other trades if power be given health officers to enforce laws designed to better the condition of factory operatives by providing better ventilation, greater cleanliness, prevention of expectoration, etc.

Tenement-house Reform.—Consumption is a house disease, and hope of stamping out this terrible scourge cannot be entertained as long as the poor of great cities are herded together in quarters so bad that the landlords would not think of stabling their horses in them. Public education may some day make such greedy owners of these wretched places of abode for hundreds of thousands of poor human beings realize that in the disease bred and fostered in such slums lies a very positive source of infection to their own families. Meanwhile, selfish greed should and must be combated by law.

Public-spirited and humane citizens emulating the example set by Mr. Henry Phipps may erect tenement buildings that will provide healthy lodgings at a minimum cost and yet bring a reasonable return to the investor. But the millennium is yet far off and meanwhile State law should bring about the destruction of these breeding places of disease and the erection in their stead of tenements that will furnish air and sunlight in proper amount.

Doubtless such measures would be decried as too radical by the landlords and by the indifferent rich, but the improved sanitary conditions that would follow would prove the wisdom and humanity of the law. The experience of England has demonstrated that a remarkable decrease in the death rate from pulmonary tuberculosis has been achieved by the tearing down of old and the erection of new sanitary tenement houses in many of her great cities.

In New York City a very gratifying and encouraging decrease (40 per cent) in the mortality from this disease has been accomplished in the past twenty years in consequence of the labors of her health officers, although working in the face of most discouraging conditions, for nowhere in the world is there worse overcrowding than in certain portions of the East Side.

Compulsory Cleansing and Disinfection of Buildings.—Another measure which is of the utmost importance to the prevention of this scourge, and which should be in the hands of the health officers, is the thorough disinfection of the premises vacated by a consumptive. A room that has been occupied by such a patient becomes a source of possible infection to any person who inhabits it subsequently. This is particularly the case when the apartment has not been properly cleaned and disinfected. If the consumptive was careless in the disposal of his sputa, the danger is doubly great.

An instance of the kind was recently brought to my notice. I saw in consultation an intelligent young Lithuanian priest who was in the second stage of pulmonary tuberculosis. As usual I inquired as to the possible source of infection and ascertained the following: His family was entirely free from the taint and he had always been healthy prior to his present ailment.

Inquiry developed the fact that for two years he had lived in a room which had been previously occupied by a man known to have consumption. This man observed no precautions whatever but expectorated about the room freely. After his removal the room had not been cleaned and disinfected, and when asked why that was the priest rejoined in effect, that it was not the habit of his nation to adopt such measures and of course he himself had not appreciated the necessity of a thorough cleaning of the quarters.

Knowing the fact that tubercle bacilli may retain their virulence for many months and even for years when permitted to remain in the dust and dirt of such apartments, and learning the family and personal history of this priest, I believe myself warranted in the declaration that all the conditions of exposure had been fulfilled in his case and that he was now suffering the consequences of the neglect of proper sanitary measures.

There are innumerable instances of houses in which this terrible disease has attacked one after another of successive tenants. Consequently it should require no arguments to prove the wisdom and necessity of administrative control in this regard. Laws should be passed requiring all proprietors of infected houses to have them thoroughly cleaned and disinfected under the supervision of the officers of health. Nay more! it should be an offense punishable by fine for such premises to be again let until after the law has been complied with.

The walls and floors should be thoroughly washed in a one to one thousand solution of bichloride of mercury or a one to thirty solution of chlorinated lime containing free chlorine, after which the room should be exposed the fumes of formaldehyde gas for from twelve to twenty-four hours. some cases the walls may have to be scraped and repapered or painted a the woodwork also painted. The expense of cleaning and disinfecting sho be borne by the department of health if necessary, but the renovation dilapidated houses should fall on the landlord. Of course selfish consid tions will arouse opposition but legislators must be made to see that pu welfare is paramount to private greed and health officials should be held responsible for the enforcement of the law.

After a room has been vacated by death or removal, the carpets, rugs and bedding should be disinfected by steam, not sulphur, by the sanitary officials and then returned to the family. The garments of the consumptive must not be worn by other members of the family or given to charity, unless having been previously boiled or disinfected in the manner stated.

Compulsory Registration.—Perhaps no one measure has excited such lively discussion and met with more opposition than compulsory notification of tuberculous cases to departments of health. The opposition comes mainly from the medical profession and various are the arguments adduced in defense of their hostility. It is urged that such a proceeding is tantamount to placing the tuberculous patient in the category of individuals dangerous to the community and must occasion unnecessary pain to the patient and his family. A consumptive already suffers enough without having the added feeling that he is an object to be shunned.

Another reason assigned for opposition to this measure is that it necessitates a violation of professional confidence since families often object to its being known that one of their members is afflicted with consumption. Still a third objection is found in the fact that Mutual Benefit Societies often refuse to pay their obligations to the families of those dead of pulmonary tuberculosis, on the ground that the insurance was obtained after the disease was known to exist. If, therefore, the attending physician reports to the health office such cases, that fact would likely invalidate the insurance.

The answer to the second objection as pointed out by Dr. Biggs is found in the fact that the notification to the department of health does not involve a betrayal of professional confidence any more than does the notification of cases of diphtheria and other contagious diseases. In reply to the first, that this measure is tantamount to placing the consumptive in the category of persons to be shunned, it may be said, if the individual comes of an intelligent family and is likely to exercise suitable precautions no further notice will be taken of the notification than to record the fact of the existence of the case. Consequently the feelings of the patient or family cannot be hurt. If, on the contrary, the person is ignorant or careless, then the notification of his case will lead to the adoption of measures of such importance to the community that they far outweigh any consideration growing out of his or his family's sensitiveness. In every such case the duty to the community is transcendent and physicians should so regard it.

The third objection seems at first glance more valid, but in reality should not be allowed to weigh against the benefits in general to accrue from notification and registration of tuberculous cases. If physicians are to be influenced by the wishes of their patrons even to the detriment of the public, then no amount of argument will avail to influence them.

The fact is that this measure is so fundamental that unless it be carried out no adequate prevention of this disease can ever be attained. If this and other measures are adopted, then in time there will be no need or excuse for the refusal to pay death-benefits on the ground that insurance was wrong-fully obtained. Meanwhile public morality and safety should be considered, and physicians who adhere to principle will find that in the end they gain more than by truckling to the dishonest motives of their patrons.

If compulsory notification of these cases is to be effectual there must be early diagnosis before the process has advanced to such a degree that germs have already been scattered abroad. To facilitate such early recognition of tuberculosis the community and the State must establish stations to which specimens of sputum may be sent for examination free of expense to the doctor or patient. In New York City there are 200 such substations, and in a single year 11,000 specimens of sputum were examined and reported on.

The benefits resulting from such a proceeding are incalculable and are not limited to mere notification of the individual case. The benefit to the patient is immense, since it entails a recognition of the real nature of his ailment at a time when appropriate treatment can be begun with a reasonable prospect of arrest of his disease. This consideration alone should win adherents to this measure both among the profession and the laity.

State Control of the Tuberculous Individual Himself.—Here we come to the vital question of how far the State has a right to restrict the actions of the consumptive for the purpose of protecting the community and ultimately ridding the earth of this scourge. For my part, I am in sympathy with all movements and all laws having so beneficent an end in view.

In Norway there are district health commissioners, whose duty it is to care for the tuberculous poor, to whom all cases must be reported and who may assign physicians to aid in ministering to these unfortunates. Still further, these commissioners are expected to see that existing laws are obeyed. According to these laws the consumptive is forbidden from engaging in any occupation which involves the handling of foodstuffs, nursing the sick or the care of children. If any person afflicted with tuberculosis is unwilling or unable to comply with the law in these respects, the commission is then empowered to remove him to a hospital or other institution where he can be appropriately treated and controlled.

Although such provisions of law may seem to bear heavily on the few, they yet have a wise and humane purpose, and it is to be hoped that the day will yet dawn in the United States when State control of consumptives will exist even as in Norway.

The Establishment of Hospitals and Sanatoria.—Such a degree of administrative control as has been advocated would and should necessitate the establishment and maintenance by the community or State of institutions designed after the most approved principles, to which might be removed all tuberculous persons who endanger the public health, either through ignorance
vicious disregard of the law. We now have asylums for the care of the insa
almshouses for the indigent and the infirm. Why should not the State
required to have institutions for the care and treatment of its tubercul
citizens who are a greater menace to the public than are the insane? F
thermore, why should there not be laws requiring all consumptives who h
no friends to care for them, adequately, to enter some such public institut
whenever this seems necessary to the officers of health?

Such measures would be found to prove a boon to the sick of this disease and would aid wonderfully in restricting its spread. In fact the adoption of these and other preventive measures would in a short time so lessen tuberculosis that it would cease to prove a menace and a burden to the State. That such a conclusion is not chimerical is shown by the reduction that has already been accomplished in the mortality records from tuberculosis, in spite of the fact that preventive measures are not as complete as it is hoped they may some day become.

Measures for Prevention in Individual Instances.—These must for the most part begin in infancy and must be based on the recognition of the two leading sources of infection, namely, cows' milk and the sputa of consumptive members of the same household as the child. Precautions to be exercised for the protection of older children and adults will become apparent or may be deduced from what has been said already, or will be said in the following pages. If the public measures herein advocated and the advice given for the protection of our little ones could only be put in effective operation there would soon be no occasion for apprehension as regards either infants or grown persons.

Let me premise what is about to be elaborated by the emphatic declaration that *if pulmonary tuberculosis is to be prevented either in the family or at large it must be by the abandonment of the old dominant notion of an inherited diathesis and the substitution therefor of the vital belief in infection.* Barring the extremely rare instances of congenital transmission of the disease from mother to fœtus, it may be affirmed that every case of tuberculosis was at one time preventable and should have been prevented.

It would be a work of supererogation to repeat what has been stated already concerning the infectiousness of milk from tuberculous cows. It is only advisable to remind the reader that although von Behring may exaggerate the danger to be attached to contaminated milk as an article of food for young infants, still the possibility of infection by this source is too great to warrant its being ignored or held in disdain. Tuberculous cattle are a menace to society and laws for their detection and death are salutary as has been shown in Denmark where thanks to strict enforcement of such provisions, tuberculosis has been almost eradicated from the dairy herds.

Care as Regards the Milk-supply.—In the United States and Great Britain the condition of the milch cattle is far less satisfactory. It is difficult to say what percentage of the cows is tuberculous, but certainly such a proportion of them as to make it incumbent on the physician to select most carefully the milk that is to be given to bottle-fed infants. In many of our large cities, e. g., Chicago, it is possible to obtain certified milk, that is, milk from herds known to be free from disease. When, therefore, this pure milk can be procured it should always be ordered. When for any reason such milk cannot be obtained the question arises, Shall the supply be had from a single cow or from a reasonably safe herd or dairy? In reply it should be stated that unless the cow has been tested and thus found to be healthy it is better to get the milk from a herd or dairy. The objections to the use of a single cow are two.

(1) It is impossible to determine absolutely by a cow's appearance that

she is not tuberculous. When far advanced the disease may show its presence by the rough coat and generally unhealthy look of the animal, and yet such is not invariable.

The truth of this statement was forcibly impressed upon me by an instance narrated to me a few years ago by the Illinois State Veterinary. A friend of his, a physician residing in a country town, found it necessary to rear his newly born baby on the bottle and therefore purchased a young perfectly healthy looking heifer. The veterinary surgeon was requested to inspect the animal, which he did. Although she looked healthy and in fine condition he suggested a tuberculin test, to which the physician consented. Before making the test, however, a number of experienced dairymen were invited to examine the cow. To a man, they declared her perfectly healthy and signified their willingness to buy her.

The tuberculin was then administered and, to the surprise of all except the veterinary, the animal reacted decidedly. She was then killed and examined *post mortem*, in the presence of the men who had declared their belief in her freedom from tuberculosis. Their astonishment can be imagined when she was discovered to be "riddled with tuberculosis." The above instance emphasizes most strongly the wisdom, nay necessity, of a tuberculin test whenever a single cow is to be selected for infant feeding.

(2) The other objection to the milk from a single and untested cow lies in the consideration that if she is diseased the entire supply, i. e., 100 per cent of the milk is contaminated. Whereas, if the milk be obtained from a large herd or dairy it is presumable that the majority of the animals yielding the supply are not tuberculous. In other words, granting that so high a percentage as twenty be diseased, but 20 per cent or one-fifth of their combined milk is likely to be contaminated, and hence the chances of the infant's taking in bacilli are proportionately fewer. The milk is not safe and yet it is in a measure safer than that from a single cow.

After all, the only safe thing to do in the case of a baby condemned to depend upon some other source for its nourishment than its mother is either a healthy wet nurse, milk from a cow that has been proven healthy by the tuberculin test, or some one of the prepared foods in the market. The sterilization of milk is now generally recognized to be deleterious.

Care against the Infant's Exposure to Bacilli of Human Origin.—This is not always possible but there are certain palpable dangers which can and should be avoided. It has been pointed out in previous pages that a young child may and probably often does become infected through tubercle bacilli ingested or inhaled as it plays on the floor, or thrusts its dirty playthings and fingers into its mouth. It may also be infected directly by its mother, nurse or other persons.

Consider for a moment the dangers to which a baby is expose
mother, father, brother, sister or other inmate of the house is consı
A tuberculous mother whose circumstances compel her to care for
nurses or feeds it. If the mother does not actually kiss the baby
lips, which in all probability she does, her fingers are likely to be
inated and with these fingers she prepares the infant's food, han

nipple or spoon with which the baby is fed, or rubs the baby's gums. In some instances the mother may actually taste the milk from the nipple or spoon to see if it is of the right temperature or sweetness, and the next instant the infected nipple or spoon goes into the child's mouth.

In case the father or some other member of the family is tuberculous and does not exercise the most scrupulous care with his sputum it is likely that the floor of the sick-room becomes infected. The baby plays on the floor and thus imbibes the germs of future disease, or as is frequently the case is held and fondled, perchance kissed by the invalid. Friends who visit the family pick up and kiss the baby, and who takes pains to inquire if such a visitor is free from early tuberculosis?

Now the precautions to be observed grow out of the consideration of these various ways in which children may become infected or are exposed. They must be rigidly banished from the room of the consumptive whenever this is possible. When such is not to be achieved, then the invalid whoever she or he may be, must be told not to kiss the child and why. He must be told to destroy his sputa in the manner to be subsequently detailed and he must take great care not to cough in the child's face but to cover his mouth with a handkerchief or cloth for the purpose of intercepting the impalpable sputum or spray which disseminates bacilli in the atmosphere of the apartment.

As a matter of fact the only means of really protecting the child is by not allowing it to enter the sick-chamber or to be handled by the invalid. Every person who has any care of the child must be made to wash his hands thoroughly before taking it up or feeding it. Furthermore, the baby should not be allowed to play on the floor of a house in which a consumptive resides, and the child's hands and nails should be kept as clean as possible. *Finally, kissing a child on the mouth should not be permitted.*

No matter how careful one may be in observing these and other precautions, it is very probable that the children of the poor who live in but a few rooms will take bacilli into their systems either by ingestion or inhalation. This emphasizes the necessity, from a prophylactic standpoint, of the establishment of sanatoria and hospitals in every community for the reception of the tuberculous poor. In the case of the wealthy it is possible to protect the children, and the consumptive may be kept at home provided he and the doctor scrupulously regard the injunctions that have been laid down. The carrying out of these precautionary measures is not to be regarded as Utopian, for I am certain that tuberculous parents will be only too glad to deny themselves the pleasure of their baby's company for the sake of their children's future welfare and protection against the misery they themselves endure.

Measures for Prevention after Exposure or Presumable Infection.—Every physician who is the regular medical attendant of any family in which there has been a case of pulmonary tuberculosis should regard himself as personally responsible for the prevention of the disease in other members of the household. This applies in particular to the children who, by reason of their tender age, are especially liable to infection and cannot be expected to appreciate the necessity and wisdom of prophylactic measures.

He must not rest content with having enforced destruction of the sputum and attention to other precautions growing out of the danger inherent in the palpable and impalpable expectoration, but should personally see that the house or room is thoroughly disinfected in the manner already described. In addition he should furnish explicit instructions concerning the health of every member of the family.

The Gospel of Fresh Air and Sunshine.—The doctor should teach both by example and precept that the prime essential to freedom from tuberculosis is plenty of pure air and sunlight in the dwelling. The poor are apt to keep windows closed in the Winter-time to save fuel, whereas if they admitted more oxygen they would not be so sensitive to the cold and would find their rooms heat up more quickly.

The rich, who rejoice in large houses and many rooms, are apt to shut out the air and light by heavy draperies and fancy that if the house is aired well in the forenoon it will suffice for the balance of the day. As a matter of fact if one enters these palatial homes he perceives that the air is dead and heavy, perfumed with the fragrance of flowers it may be but hot and oppressive.

What now shall we say of the air in offices, shops, places of amusement, churches and public conveyances, in hotel lobbies and, in short, in every inclosed place where people spend their waking and working hours? We boast of our twentieth-century civilization and yet are content to breathe air unfit for the lower animals. Why do not architects devote their talents to devising means of adequately ventilating private and public buildings, and why do not health boards and physicians in general devote some of their energies to instructing the people, both educated and ignorant, regarding the benefits of fresh air?

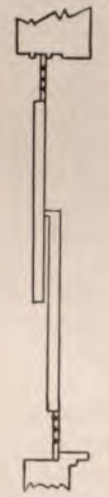


FIG. 73.—Method of window ventilation.

The doctor should teach the families under his care that in their homes they must *keep windows open day and night.* It is not necessary to have a draught, for by means of a screen or device about to be described fresh air can be admitted in abundance yet without endangering the inmates.

There are several excellent *ways of admitting fresh air* without causing a draught, as follows: The window may be dropped four inches and a board inserted in the opening thus created (Fig. 73) which board is punctured with numerous funnel-shaped openings whose broad extremity is on the inside of the board. This lowering of the upper sash creates a crack between the two sashes which admits a stream of fresh air from the outside. The perforations in the top board serve as a means of escape for the hot foul air that rises to the ceiling. In this manner a room may be ventilated fairly well, and if necessary two windows on opposite sides of the apartment may be similarly arranged. In the Summer, and on warm days at other seasons, one or more windows should be kept wide open all the time.

In some cases it may be well to raise the lower sash as well as drop the upper one and then insert a perforated board at both top and bottom of the window. The funnel-shaped openings must be reversed however so as to allow the entrance of air at the bottom and effect the escape of air at the top. In the lower board therefore the perforations decrease in diameter from without inward so that on the room-side of the board they show an opening of only one-quarter inch while in the top board the perforations point outward. Since these openings should not be large, they must be thickly studded over each board, yet not so closely as materially to weaken the board, which may be of pine and about an inch in thickness.

Another contrivance by which an abundance of pure air may be supplied is the following: The bottom sash is raised about a foot. On the room-side of the window and in front of the opening a board or plate of glass is placed in such a manner (Fig. 74) that it slants from above downward and outward. In other words, the bottom edge of the board which is held in position by cleats at each side, rests on the window-sill close to its outer edge and slants from this point inward and upward so that its upper margin is separated several inches from the lower edge of the open sash. The board should reach an inch or two above the level of the bottom of the sash. Arranged in this position the board permits a stream of air to enter beneath the open window but deflects the current upward toward the ceiling and thus prevents cold air from blowing in directly on the occupants of the room.

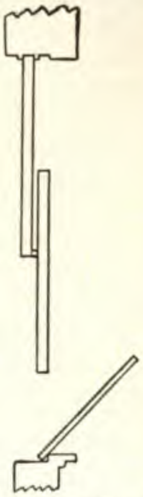


Fig. 74.—Method of window ventilation.

Still another means of air-supply without unpleasant draught is by the insertion into the open window of a wooden frame which is covered by a single thickness of cheesecloth or very thin muslin. An ordinary wire screen used to keep out flies and other insects may be covered by a single layer of cheesecloth or cotton mosquito netting. This permits a good deal of air to enter and yet serves to break somewhat the force of the wind, so that if the bed is not directly in front of the window the inmate of the room will be sufficiently protected.

It is particularly in the children's nurseries and in the bedchambers at night that plenty of fresh air must be insisted upon. People have a silly prejudice against night-air, fancying it is damp and unhealthful. It may be damper and certainly is colder than that of the day when the sun is shining, but it should be purer because not so contaminated by dust stirred up by passing vehicles and by the smoke of factories.

At all events, air from outside the house is purer and freer from germs than is that within. For this reason every physician should preach the gospel of fresh air to his patients and friends. If a bedchamber is small and it is impossible to place the bed so as to avoid a direct draught, then the next best thing is to ventilate through an adjoining apartment or the open door. Do not, however, let the family commit the error of fancying that it is enough to open all the bedrooms into a common hall or sitting room without having an abundance of fresh air admitted to these latter. The low temperature of a Winter night should be no barrier to the carrying out of this fresh-air supply. By means of plenty of bedclothes, and sleeping-bags for the young children, the sleepers can be kept warm.

The *necessity for sunshine* is equally important, since the sun's rays are a powerful germicide. Unfortunately, the poor and even the well-to-do dwellers in cities cannot always command a supply of sunshine in their homes. There is no excuse for its lack in the country and small towns, however. The value of sunshine and the harmfulness of shade and consequent dampness were shown by Henry Y. Bowditch many years ago. He proved a direct relationship to exist between shade and dampness in house and soil and the prevalence of pulmonary tuberculosis. Therefore, let us insist on the beneficence of sunshine in our homes and in particular in our bedrooms and nurseries.

For persons who have been exposed to infection and evince signs of low vitality there is no mode of obtaining fresh air equal to *sleeping out of doors* as will be described under Treatment. Even in the congested tenement districts of great cities something can be accomplished in this direction if roofs and balconies are utilized. As for the prosperous there should be no insurmountable obstacle to the accomplishment of this highly salutary measure. In the country there can be no valid excuse for the failure to carry it out.

Measures for improving the general health and thereby increasing tissue-resistance must include all those resources with which bountiful nature has provided us but which we are prone to neglect for drug therapy. Delicate children of the better class should be reared in the country or in some suburb where play can be had outdoors and sunshine can be obtained. As they grow older they can be encouraged to spend their Summers in the forest or on the shores of some lake where game and fish abound. Life in the woods is invigorating and conduces to healthfulness of mind as well as body.

Those condemned to remain in the city can spend as much time as possible in the parks and playgrounds with which our American cities are provided. They can be taught such breathing and other exercises as will develop their chests, invigorate circulation, stimulate excretion of waste products and strengthen the general muscular system. Young persons inclined to spend too much time indoors reading, practicing on some musical instrument, sewing, etc., must, if circumstances permit, be made to get outside for wholesome recreation and pure air. Their appetites will be strengthened and their assimilation improved.

Another very useful measure for invigoration of the body is a *cold tub or rub* in the morning. Care must be taken to insure a prompt and good reaction for otherwise the early morning bath will not be followed by a sense of well-being. I am of the opinion that there are few if any even delicate chi or young adults who cannot become accustomed to this means of invigo the circulation in the skin and lessening the sensitiveness to cold.

The *dietary* is a very important matter and must be directed c and not by the vague general statement to eat all the good food possibl attempt should be made to get children to eat all kinds of food that is some and well cooked. In particular they should be induced to take fa gravies, butter and cream. Indeed, I regard it as a great pity when they are allowed to grow up with an aversion to milk and many kinds of vegetables. It is equally unfortunate when delicate girls restrict themselves to fruits, cereals and a few daintily prepared articles to the exclusion almost, of milk, eggs and meats which provide the proteids so essential if one is to overcome latent tuberculous infection.

The chloro-anæmia so common in young women who are suspected of latent infection is more surely overcome by plenty of fresh air, sunshine and good nourishing food than by all the iron and other tonics that can be poured down them. The roses will return to their cheeks and brightness to their eyes when they live outdoors and get close to mother nature.

The keynote of the prevention of pulmonary tuberculosis is sounded by von Behring when he declares that the bacillus taken into the system in early infancy lies dormant until fresh infection occurs or tissue resistance is lowered through puberty or some other condition which makes a drain on vitality. Accordingly, young persons with a tuberculous family history should be taught to do all they can to maintain their health and to avoid whatever may undermine their strength.

If they are compelled by force of circumstances to work in factories, shops, offices and the like they must do what they can by wholesome food and as many *leisure hours* as possible in the parks or country to counteract the depressing influence of their occupations. In particular should they avoid contact with friends or relatives suffering from consumption. A young woman of a tuberculous family who contemplates marriage should be told of the peril of frequent childbearing and prolonged lactation. In the event of her marriage she should be kept under the watchful supervision of a physician.

Unfortunately, there are many of the very poor who are doomed to this disease, for, as some one has said, "the destruction of the poor is their poverty." Those, on the other hand, whose means, although restricted, yet permit a choice of residence should be told to select their dwellings with a view to the maximum amount of fresh air and sunshine. If at all practicable their residence should be in the outskirts rather than in the congested wards of a large city.

The young man who has already lost some near relative from consumption and hence may be said to have a tendency to the disease, and who is falling into bad habits is in very great danger. He should be warned by his family doctor that if he continues to dissipate he will surely prepare the soil for the growth of the germs presumably already in his system. In particular must he be warned against *alcohol,* since its abuse, which is but too common in all classes, furnishes a powerful predisposing factor in the causation of pulmonary tuberculosis.

Cigarette smoking, too, is highly injurious to the growing lad who may be said to have weak lungs or, in other words, has been exposed to infection. The inhalation of tobacco smoke irritates the epithelium of the bronchial passages besides proving deleterious to the general health. If, in addition, the young man spends his evenings in a barroom and deprives himself of

needed sleep he must not be surprised if in time he falls a prey to his f
foe, consumption.

This subject of individual prevention against pulmonary tuberculosis
be extended almost indefinitely, certainly to a wearisome length. Enoug
been said to indicate the measures to be carried out by the physician an
individual concerned. The weapons to be employed are pure air, sun
wholesome food and clean living in every sense. It must be borne in
however, that the public will not employ these weapons unless taught
so by physicians. Prophylaxis is far higher and nobler than therapy
the community has a right to demand of its physicians that they instru
publicly and privately in the principles of prevention of this plague that c
off the very flower of its youth and manhood.

CHAPTER XXX

TREATMENT OF PULMONARY TUBERCULOSIS

If the reader expects or desires the advocacy of some specific medication or a long list of drugs which he can pour down the throats of his confiding but misguided patients, he will be disappointed. Modern medicine has passed beyond or rather is rapidly passing out of the age of expectorants, tonics and even cod-liver oil into that of hygiene and diet. We are prone to think of this as a new departure in the management of tuberculosis, whereas it is a modification and elaboration of a plan of treatment practiced by Sydenham in the middle of the seventeenth century and advocated by Bodington in 1830.

The teachings of these two pioneers in the rational management of this disease did not impress the profession at large, perhaps because not widely known, and hence the credit must be given to Germany of having initiated this new movement. To Brehmer at Gerbersdorf in the mountains of middle Germany and to Dettweiler in the Taunus range of southern Germany are due the inauguration and establishment of what has revolutionized the treatment of consumption.

The Modern Sanatorium Treatment.—As implied by its name this is a definite system of management having as its fundamental principle the removal of the tuberculous to an institution especially designed and constructed to carry out an open-air treatment combined with proper hygiene and diet. Such institutions are sometimes spoken of as Closed Sanatoria, not to signify a place where patients are shut in but to distinguish them from health resorts or open sanatoria where all the invalids do not live in the same building or set of buildings.

The advantages of this mode of management are many and the results obtained in suitable cases are so conspicuous that the number of sanatoria is rapidly increasing in Europe and North America and, so far as I know, throughout the civilized world. I have said the results obtained in suitable cases. By this, it is not meant to imply that the sanatorium treatment is not adaptable to all cases, but merely that, as a rule, advanced cases are not received in such institutions, for the reason that, as they are generally hopeless under any form of management, it is thought more humane to reserve the limited accommodations for early and presumably curable cases. Institutions to which persons with advanced consumption are admitted are called hospitals, but in them the same principles of management are carried out with such modifications as the condition of the inmates necessitates.

It is not proposed to enter minutely into a description of model sanatoria, since this may be found in such books as Ransom's and Walter's, but merely to state such facts as are deemed essential to an understanding of the modern sanatorium treatment of pulmonary tuberculosis. Such an understanding is requisite if one will intelligently carry out the principles in the care of tuberculous invalids at their homes. This is now being attempted in all parts of the world and in all climates, while in so-called health resorts it forms a prominent feature of phthisiotherapy and is the main argument in favor of sending consumptives away from their homes.

Location of the Sanatorium.—Many institutions in Germany and other parts of Europe, and for that matter in the United States, are situated in the mountains, but that such a situation is not indispensable is proven by the results obtained at sanatoria built on the plains. Wherever the institution is erected, it must be sheltered from north and east winds by wooded hills or a forest. If possible the trees serving as a wind-break should be pines and firs since the air in their vicinity contains ozone.

The *soil* on which the sanatorium stands should be porous so as to dry out rapidly after a rain. The sandy nature of the ground on which pine trees grow furnishes an additional reason for the location of the sanatorium in the shelter of such forests. The free drainage provided by the slope of a mountain or large hill also recommends such a situation.

Sunshine as well as pure air is a great desideratum and hence the building should be open to the south and west if possible but at all events to the south. It should not be near enough to a city or factory to have its air-supply contaminated by dust or smoke. On this account, therefore, a sandy arid plain would be objectionable because permitting strong winds that blow over the country to sweep clouds of dust to the lungs of the invalids as they recline or stroll out of doors.

It is very desirable, moreover, that there be a *pleasing and diversified scenery* since nothing is more wearisome to the person doomed to pass months at a sanatorium than a monotonous uninteresting prospect. When pleasing views are not obtainable, then the want should be supplied by gardens, shrubbery, etc., and a well-kept stretch of green turf. The grounds should be extensive and provided with pleasant shady graveled walks that, winding in and out among the trees, diversify the scenery and tempt those to walk who are able to do so.

Resting-places (*liegehallen*) form a notable and indispensable feature of all properly designed sanatoria. They must be ample for the needs of the whole number of patients at any one time, and must permit the maximum of pure fresh air without undue exposure to wind, storm and sunshine without the discomfort of direct exposure to the sun's rays in the hot days of Summer. They should also be elevated above the ground. Some authorities insist on these verandas being connected with the parlors or halls of the institution, not with the sleeping apartments. Others, recognizing the inconvenience of such an arrangement permit the bedchambers to connect by means of doors with roomy suitably protected balconies. At still other institutions there are pavilions in the grounds where patients can rest in the open air, and some of these pavilions are made so as to rotate and thus turn their open side in any direction according to wind and sun. This latter provision is not necessary, however, if the sheds or pavilions are properly constructed.

The ventilation of the bedrooms, halls, living rooms, etc., must be as perfect as architectural skill can make it. The various apartments must be homelike and attractive yet not hung with heavy curtains or carpeted. Indeed, there should be a minimum of rugs and other articles of adornment on which dust and germs may collect. In short, the sanitary arrangement of the building must be up to date in every particular.

The bedrooms must be spacious, at least 1,500 cubic feet, well ventilated and so situated as to receive sunshine sometime during the day. For this reason they cannot be on the north or east side of the building and if they open into private balconies where the patients can take the air this furnishes an additional reason against their having a north or east exposure. Moreover, each balcony must be provided with screens or canvas curtains for the purpose of affording protection against storm or the direct rays of the sun in hot weather. Such protections are also necessary on the public veranda opening off the ground floor.

If the patients take their meals in a common dining hall, this must be also commodious, well ventilated and exposed to the rays of the sun. In short, the institution must be carefully planned with a view to the greatest possible amount of fresh air and sunshine. As this is difficult if not impossible in a single building, some advocate the so-called Cottage Sanatorium like that at Saranac Lake. Even when the invalids are not all accommodated in cottages holding from four to eight persons, it will generally be found that a number of such small houses are grouped in the vicinity of the main hall.

The administration offices should be located in or closely connected with the main building, and in a well-equipped institution these will be provided with every appliance necessary for the examination and treatment of the patients. The majority of the medical staff and nurses may occupy a separate building but at least one physician and one or more attendants should sleep in the sanatorium proper so as to respond without delay to the call of any patient.

The dietary must be well prepared, inviting and determined with special reference to the needs of the tuberculous. The principles of such a dietary will be considered under that heading. Physicians and patients generally eat together, an arrangement that enables the medical staff to keep track of the amount the patients consume and to encourage those who are inclined to neglect their food.

In many sanatoria, particularly those in Germany, ample facilities are afforded for the carrying out of hydrotherapy on which much stress is laid. This includes douches, sponge baths, cold wet rubs, etc. Patients whose strength permits receive this cold-water treatment in bathrooms.

The *régime* on which is based the modern sanatorium treatment must be carefully planned and carried out systematically. Nothing is permitted to go haphazard and every inmate must conform strictly to the rules of the institution. *In nothing is more pains shown than in the collection and destruction of the sputa.* Not only are the inmates required to carry with them constantly

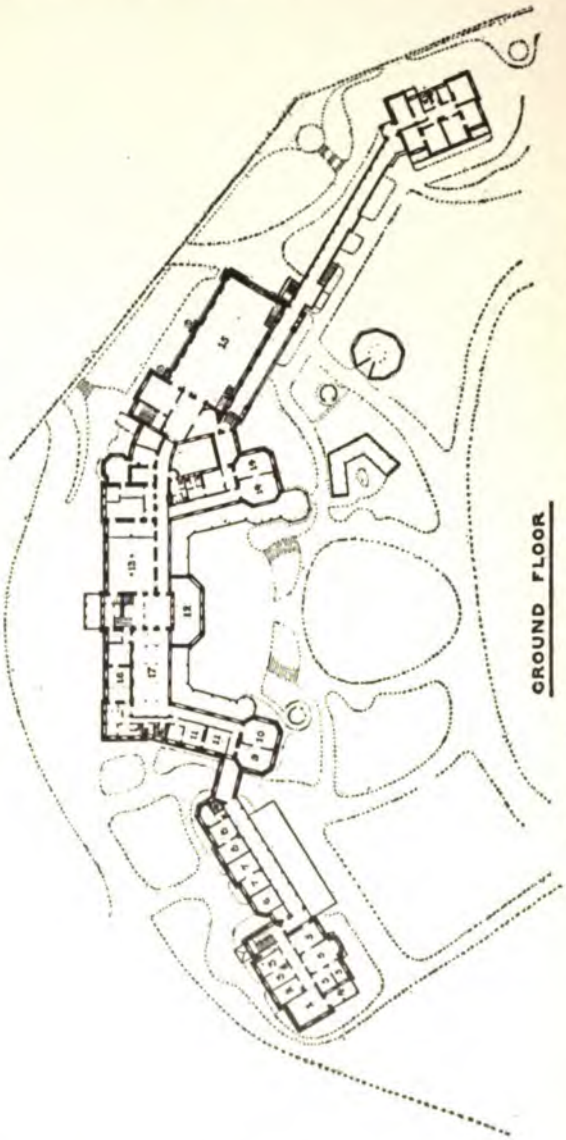


FIG. 75.—THE FALKENSTEIN SANATORIUM—GROUND PLAN. (WALTERS.)

1. Board room.
2. Visitors' room.
3. Rooms for the staff.
4. Veranda.
5. Consulting and waiting rooms and laboratory.
6. Lavatory.
7. Mortuary.
8. Gardeners' quarters.
9. Library.
10. Billiard room.
11. Visitors' rooms.
12. Winter garden.
13. Reading room.
14. Conversation and music rooms.
15. Dining saloon.
16. Office.
17. Winter garden.

suitable sputum-cups but throughout the institution are provided cus
which are so placed or so constructed as to make the spilling of their co
impossible whenever by accident one is overturned. The receptacles
larly emptied, disinfected and their contents carefully destroyed. E

caution possible is taken to guard against the dissemination of bacilli through the air of the apartments (Fig. 75).

The foregoing description makes plain the fact that if the modern sanatorium is to fulfill the purpose for which it is intended it must afford every facility for the carrying out of the open-air treatment of pulmonary tuberculosis. Inasmuch as this cannot be accomplished within four walls the institution must be designed and conducted with a view to life in the open air in the easiest, safest and most agreeable manner. It is realized that attempts to combat tuberculosis by means of drugs fail signally, and therefore the one dominating purpose must be the increase of powers of resistance. Everybody concerned with the management of the institution must be actuated by this motive and every individual admitted for treatment must be imbued with this idea so as to coöperate intelligently in its attainment.

The results achieved in closed sanatoria are stated in figures since general statements unsupported by figures would not be accepted. Statistics are not entirely satisfactory however and do not always express the condition of the patients from which the tables are compiled. It is difficult to estimate results for many reasons and, to my mind, the best proof of results is to be found in the steadily growing popularity of sanatoria for this class of sufferers throughout the civilized portions of the globe.

Methods of estimating results are not uniform, the degree of general improvement being taken by some and by others the change for the better in the local process. The phraseology employed to express the individual's condition at time of discharge differs, since by some the terms cured, nearly cured, improved, stationary and worse are used, while others content themselves with better, stationary and worse. The class of patients received and the stage of the disease on admittance also are not the same in all institutions, and lastly the individual factor on the part of the reporter must be taken into account.

On the whole, the most satisfactory basis for the estimation of results is found in the working capacity of the person and it is on this that the reports from most of the sanatoria for the working classes in Germany are based. As might be expected, the results bear a tolerably direct relationship to the financial status of the patient as well as to the stage of his disease, since persons not dependent on their own exertions for their support are able to avail themselves of sanatorium treatment for a long or for possibly an indefinite time. Consequently, if the disease is taken properly in hand in its incipiency it is susceptible of cure in probably 80 per cent of cases, while in the second and third stages the prospect of arrest grows progressively less even in persons who can command every facility necessary.

In his address, History of the Tuberculosis Work at Saranac Lake, New York, delivered before the Henry Phipps Institute at Philadelphia in October, 1903, Dr. E. L. Trudeau gave the following figures for 1902: 165 cases were admitted in all stages of the disease and of the 40 really incipient cases 75 per cent were discharged as apparently cured, 15 per cent arrested and 10 per cent were improved. Of 99 advanced cases 12 per cent were discharged apparently cured, 57 per cent arrested, 22 per cent improved. The subjoined tables give the results in four of the People's Sanatoria of Germany. They are copied from the report of the Imperial Bureau of Health, 1904.

People's Sanatorium of Planegg.—For Male Patients only.

(Number of patients treated, 845.) Discharged:

Cured.	Almost cured.	Much improved.	Improved.	But little improved.	Not improved.	Worse.	Died.	Not specified.
2=0.2%	31=3.7%	50=5.9%	465=55.0%	136=16.1%	108=12.8%	50=5.9%	3=0.4%	24=2.8%

(Number of patients treated, 842.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specified.
407 = 48.3%	16 = 1.9%	259 = 30.8%	157 = 18.6%	3 = 0.4%	27 = 3.1%

People's Sanatorium of Grabowsee.—For Male Patients only.

(Number of patients treated, 2,322.) Discharged:

Cured.	Improved.	Not improved.	Worse.	Died.	Not specified.
105 = 4.5%	2,055 = 88.5%	80 = 3.4%	70 = 3.0%	12 = 0.5%	11 = 0.5%

(Number of patients treated, 2,316.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specified.
1,156 = 49.9%	336 = 14.5%	610 = 26.3%	202 = 8.7%	12 = 0.5%	17 = 0.7

People's Sanatorium of Ruppertshain.—Division for Male Patients.

(Number of patients treated, 1,319.) Discharged:

Cured.	Improved.	Not improved.	Worse.	Died.	Not specified.
114 = 8.6%	1,114 = 84.5%	76 = 5.8%	13 = 1.0%	2 = 0.2%	20 = 1.5%

(Number of patients treated, 1,309.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specifie
880 = 67.2%	38 = 2.9%	277 = 21.2%	112 = 8.6%	2 = 0.2%	30 =

People's Sanatorium of Ruppertshain.—Division for Female Patients.

(Number of patients treated, 480.) Discharged:

Cured.	Improved.	Not improved.	Worse.	Died.	Not specified.
69 = 14.4%	356 = 74.2%	48 = 10.0%	7 = 1.5%	....	5 = 1.0%

(Number of patients treated, 475.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specified.
308 = 64.8%	5 = 1.1%	89 = 18.7%	73 = 15.4%	....	10 = 2.1%

Dr. Weicker's Sanatorium in Görbersdorf (Krankenheim).—Division for Male Patients.

(Number of patients treated, 2,660.) Discharged:

Cured.	Improved.	Not improved.	Worse.	Died.	Not specified.
15 = 0.6%	2,318 = 87.1%	218 = 8.2%	89 = 3.4%	20 = 0.8%	25

(Number of patients treated, 2,660.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specified.
1,999 = 75.2%	58 = 2.2%	264 = 9.9%	319 = 12.0%	20 = 0.8%	25

Dr. Weicker's Sanatorium in Görbersdorf (Krankenheim).—Division for Female Patients.

(Number of patients treated, 1,359.) Discharged:

Cured.	Improved.	Not improved.	Worse.	Died.	Not specified.
2 = 0.1%	1,148 = 84.5%	144 = 10.6%	61 = 4.5%	4 = 0.3%	....

(Number of patients treated, 1,359.) Discharged:

Fully able to work at former occupation.	Fully able to work at another occupation.	Partly able to work.	Not able to work.	Died.	Not specified.
986 = 72.6%	48 = 3.5%	141 = 10.4%	180 = 13.2%	4 = 0.3%	....

Home Treatment Based on Modern Sanatorium Methods.—The results obtained in closed institutions are so striking that a general movement is everywhere apparent toward the application of these principles to the management of tuberculous patients at health resorts and in the home. This is one of the encouraging signs of the times and, taken in connection with the efforts being made for prevention gives promise of a day when consumption will no longer be the dreadful scourge it now is.

In spite of the numerous contributions to this subject which have appeared and are still appearing in medical literature there is a lamentable lack of knowledge among medical practitioners, not alone concerning the methods of putting this treatment into effect at home but also regarding its principles. It is hoped, therefore, that these pages may aid in spreading this knowledge among the profession.

Applicability of the Treatment.—In the outset let it be understood that the hygienic and dietetic management of pulmonary tuberculosis is applicable to all cases though not to the same extent nor in the same manner in all. Tuberculosis, even when it affects the lungs, is a distinctly curable disease but the ease and certainty of a cure are largely governed by the stage and extent of the pulmonary changes. In strictly incipient cases a cure may be attained in something like 80 per cent, whereas when mixed infection has been added and the pulmonary disease has become consumption the affair assumes a very different aspect. Therefore, if this plan of treatment is to accomplish satisfactory results it must be begun early, and the earlier, the better. For this reason the practitioner must perfect himself in diagnosis by physical examination.

Means of Securing Life in the Open Air.—This is the fundamental proposition, the goal toward which the phthisiotherapist strives. It must never be lost sight of, and attempts at its attainment must never be abandoned until circumstances prove it wholly impossible. The only obstacle in the way of its accomplishment in early cases is the financial status or the environment of the invalid. He who is not compelled to earn his daily bread must abandon his ordinary pursuits if these are indoors and spend his entire days and if possible his nights likewise in the open air. If he resides in a city he must betake himself to the country but if his home is in a town and is surrounded by a yard he may find the necessary space there. An inexpensive shack or properly constructed tent may be erected in the yard or a hanging balcony or a veranda may be utilized. The latter are less conspicuous and often can be made to answer the needs as well as a shelter in the yard.

Tents are for the most part very objectionable because made of canvas which, being waterproof, is also impervious to the air and is unprovided with means of adequate ventilation except through the flap that serves as a d way. A circulation of air within a tent or apartment is necessary to ven tion and unless a tent is provided with some means for air-circulation worse than a room in a house. Accordingly the ordinary army tent is a c sion and a snare and this cannot be too strongly emphasized.

The only tents I would venture even to recommend are the Gar Sanitary Tent and the Tucker Tent. The former, shown in Fig. 76, designed by Dr. C. F. Gardiner of Colorado Springs and was inhabited by him for a number of months in the midst of Winter. It is made either of white or dark khaki or of 12 or 15 ounce duck, stretched over a six-sided framework of wood without any center pole, without stakes or guy ropes, so that it stands firm like a house. This framework can also be made of jointed gas pipe. It has a raised floor made of matched boards and in a single section or several, as desired. The lower edge of the tent-wall is fastened several inches below and one inch from the side of the floor all around, so as to insure an influx of air that is gradual and without draught. At the apex of the tent is an opening properly covered with a zinc cone against storms which provides the necessary exit for foul air. The tent is heated by a stove placed in the center of the floor, the stovepipe passing through the wall of the tent near the top. This tent is believed to provide perfect automatic ventilation. For additional particulars see Dr. Gardiner's little book The Care of the Consumptive. Also County and City Care of Consumptives; Some methods of Housing, published by the Committee on the Prevention of Tuberculosis of the Charity Organization Society of the City of New York, 1905.

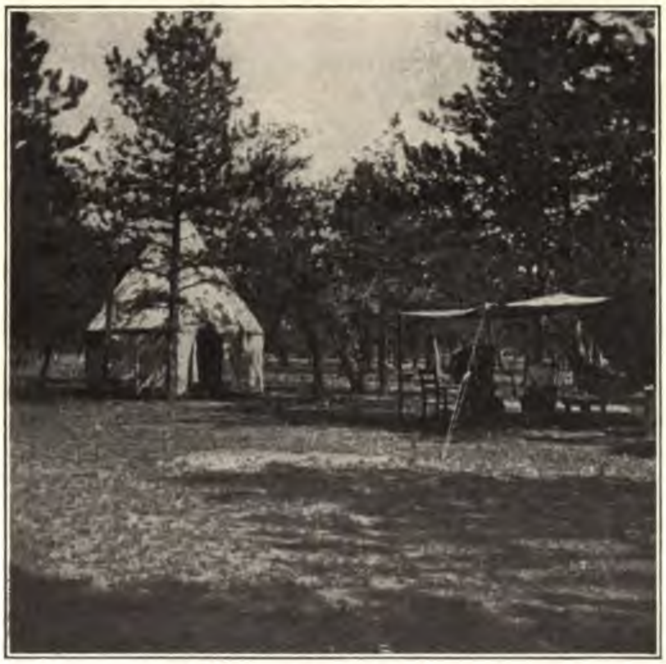


FIG. 76.—The Gardiner Tent.

The Tucker Tent, extensively used at the Association Health Farms of the Y. M. C. A. of Denver, is shown in Fig. 77. It is ventilated by means of a combination roof and fly ventilator with regulator attachments placed in the center of the top of the tent and by means also of two awning frames which constitute the front wall and can be raised at various angles so as to serve as awnings. These can be removed entirely if desired. The side walls are so constructed as to create an adjustable opening which may be widened at pleasure and thus secure further ventilation without draught. In addition there is a flap of canvas covering over the inside roof so as to permit the circulation of air above the roof, thus acting as an air-space to secure coolness within the tent. There are also a door and two six-light windows all protected by wire netting. It also has a raised floor.

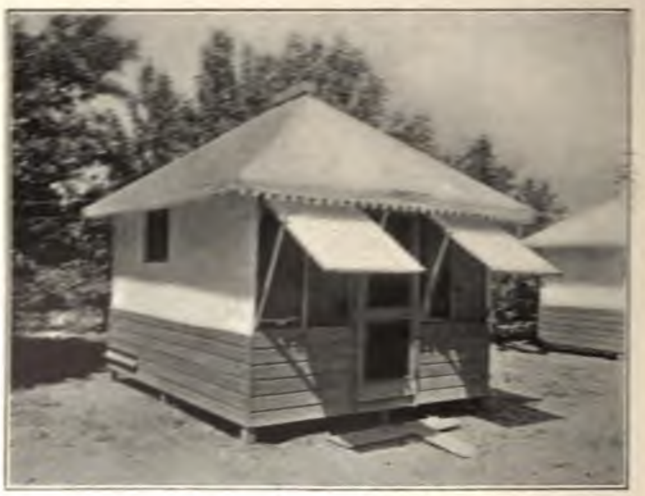


Fig. 77.—The Tucker Tent.

It is my belief that even these tents are less desirable than would be a cheap shed or shack constructed so as to provide plenty of pure fresh air and having an elevated floor. After all, any system of ventilation is less effectual than is desirable for the sojourn of one who wishes to live in the open air and yet obtain necessary protection from the elements. On this account the best shelter is a rough, cheap shack or lean-to made of boards or of boards and canvas, one side of which at least can be kept constantly open. In Fig. 78 is shown the kind of lean-to designed by Dr. A. M. King, the medical director of the Loomis Sanatorium at Liberty, N. Y.

As originally constructed the King Lean-to was capable of accommodating eight invalids. For the uses of a single individual it could be lessened in size and expense. As now improved it consists of a shed open in front, with an overhanging roof and with the ends so constructed as to be opened or closed as occasion demands. Along the front and between the stanchions canvas curtains may be suspended and so arranged as to be raised or dropped at will.

Back of this open shed on which the patient's bed or chair is to be placed is a small extension communicating with the front by a door and so constructed as to serve as a retiring or dressing room. This room must be furnished with heating and toilet arrangements so that the patient may here prepare for the night, receive his hydropathic treatment and dress in the morning. Such a shack or lean-to is a capital device for securing the open-air treatment wherever adequate yard space can be secured. It is not very expensive and can be made as cheaply or extravagantly as the finances and whim of the patient may dictate.

A shed constructed according to the principle of the King Lean-to could be cheaply made by any country carpenter out of cheap boards, or one that would answer for a temporary shelter in the Summer could be made of canvas at a not very heavy cost. A modification of the King shack can be procured of E. J. Noblett at 121st and Peoria Sts., Chicago, for $175 or less if lots of ten or more are ordered.

Whatever be the exact form or construction of any shed it must have a raised floor and be such that one side or, still better, two sides are always entirely open. By means of curtains that can be raised and lowered at will or by means of sliding sides it will be possible to keep closed as much of the shed as is necessary to protect from wind or storm no matter from what quarter these may come.

The great difficulty is that a person sensitive to cold or afraid of a little dampness will be likely to shut up too much of his shack by night or even by day. For this reason such a shack as Dr. King's, which is always open on at least one entire side, is the kind that should be kept in mind whenever one is planning a shelter for a tuberculous patient. It is on this account that a balcony or gallery proves a very good place on which a person can spend his waking and even his sleeping hours. If a hanging balcony does not already exist one can be readily built by a carpenter at a slight cost. A window looking to the south or west may be cut down to the floor and thus converted into a doorway wide enough to permit the passage of a narrow bed or

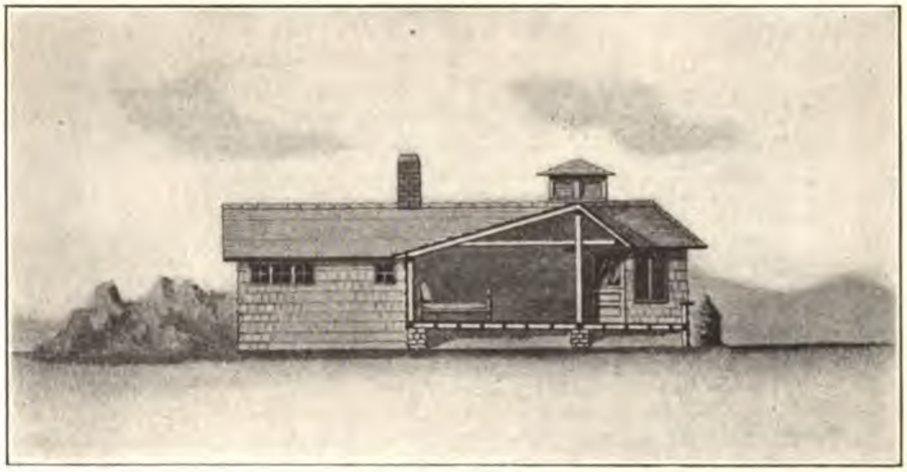


FIG. 78.—The King Lean-to.

cot. The carpenter then fastens up stout iron or wooden braces on which he constructs a balcony of sufficient width and length to afford a convenient place for the invalid to have his bed or an easy reclining chair or couch as well as a table. This balcony should not be too contracted for ease and comfort.

It should be roofed over and provided with some means of protection at the front and sides which can be raised or lowered according to the weather. A canvas awning may answer for the roof in some cases and adjustable screens may serve to keep off the wind, but some more substantial provision is necessary against heavy rains. I have frequently known a back veranda of a second story flat in a not very thickly populated portion of Chicago to be thus equipped and accommodated to the needs of a consumptive for a number of Summer and Fall months to his betterment in certain symptoms, if not to his recovery.

A country or suburban residence usually has one or more porches which can be utilized as excellent open-air resting-places for tuberculous invalids. All that is required is one of sufficient width and length, with a roof from which canvas curtains or Japanese porch-screens can be hung or which can be sheltered from wind and storm in some other way. It should be situated by preference on the south or west side of the house, and yet an east or a north exposure is far less objectionable than the remaining indoors even though the windows be kept open. It may not receive much sun but it can be so protected during stress of weather as to guard the patient from cold and wet while still leaving one portion of the piazza entirely open to pure fresh air.

A young woman in the second stage of tuberculosis of the right lung with septic symptoms spent three Fall months on a front porch having a northern exposure and not far distant from the street of her village. Not only did she not once take cold, but she so far improved that about Thanksgiving time I was able to transfer her to Colorado Springs where, conditions being more favorable, she made gratifying progress toward an arrest of her disease. The next Summer her father insisted on taking her back home and, an indiscretion being committed, she had a relapse and died.

Another of my patients whose circumstances as well as physical state prevented a change to a more salubrious climate spent his days and nights from April to Fall in a rough, cheap shed in his small back yard with the result that he not only improved in a truly surprising fashion but he could not bear to stay in the house even during the violent storms and changes of temperature that mark weather on the shores of Lake Michigan.

If there be a shady yard or a vine-clad summerhouse or arbor a hammock may be swung under a tree or in the arbor and here a person having incipient tuberculosis may while away his days in a very agreeable manner, returning to the balcony only at night or when the weather by day is unpleasant.

As previously remarked it is not difficult to devise some means of securing life in the open air for individuals who have means or a residence in country or town. The affair is far otherwise for the poor who cannot get away from the crowded district and unhygienic tenement house of the city. For such it is suggested that the plan be utilized of living and sleeping on the roof, as is done in New York City. Not only are roof-gardens and playgrounds rapidly becoming very popular in that city, but it is stated that the orthodox Jews who live in congested blocks celebrate the exodus of their race from Egypt by erecting wooden sheds on the roof, thatching them with straw and here spending the required eight days in solemn thanksgiving and other rites.

Some of the New York hospitals as Mount Sinai, the French Hospital and a new lying-in hospital are also reported to use their roofs for sun baths for their patients. If now this "top story" as it is called is made use of in such ways, why cannot tuberculous patients be given a chance for fresh air and sunshine in the same manner? I am sure that charitably minded citizens could be induced to permit the use of roofs of large buildings if health boards and physicians would only call their attention to the benefits therefrom. Practitioners having the welfare of the tuberculous poor at heart and having fertile brains could secure quite satisfactory accommodations for indigent tuberculous patients on the roof under the stars if they would only devise some inexpensive means of shelter from the too intense sun and from storms. A hint to the wise is sufficient.

Rules for the Enforcement of the Open-air Treatment.—The necessity and benefits of pure fresh air being acknowledged it now remains to lay down the rules that should govern the taking of the air. Formerly tuberculous invalids were told "exercise all you can out of doors." The result was that consumptives scarcely able to crawl about, were seen dragging their emaciated forms along the streets, into public parks and any other place where their fancy might lead them. Their fever was increased, their wasting continued and their desires were frustrated. We now know that such advice was a mistake.

The rule to-day is for the tuberculous patient to *remain at rest in the open air as long as he has a temperature above* 99° *F.* By rest is not meant sitting for a while in a chair and then, when fancy impels, to get up and stand or walk about for a time, thus merely playing at rest, but the patient who is told to rest must obey literally if he would derive the benefit desired. The person with incipient disease who still retains fair strength and weight may be permitted to recline in a suitable chair or on a couch or in a hammock. The consumptive who has mixed infection with a daily range of fever to 101.5° F. or higher must obtain his rest in bed the same as would a typhoid-fever sufferer.

The chair for the incipient case may be an ordinary steamer chair or one especially constructed which fits the body. It should have a bend to fit the knees, arm-supports, a foot-rest and an adjustable back high enough to support the head. It should be cushioned so as to be soft and yet must not be too warm in the heat of Summer. There may be an adjustable shelf so attached as to be swung around in front to serve as a support for dishes, for a book or for writing implements, for such as are allowed to divert themselves by reading or writing.

Persons more ill should be kept in a cot or bed and must not be permitted to make any exertion whatever that is likely to militate against improvement. In all cases incipient as well as advanced, there should always be plenty of warm rugs and wraps at hand for instant use in case the temperature change or the person become chilled in consequence of inaction. The strong patient may adjust this covering himself but an attendant must do so for the seriously ill. Consequently a nurse should be near by to hear the call of the invalid. Delay in this matter may readily result in a disastrous chill.

Protection against getting Chilled is a Matter of Extreme Moment.—The late Dr. Dettweiler told me that every person at his establishment was instructed to resort to some simple device to start the circulation as soon as he felt chilly in the slightest degree—to stir about in his chair or bed, to stand up and take a few steps if well enough to do so and dressed, and to adjust extra covering kept constantly at hand. If these were not enough he was to summon an attendant who applied friction to the extremities or brought a hot-water bottle and tucked him up, snug and warm. If, in spite of these precautions, warmth of body did not speedily return the patient was taken to his room and further measures were tried. If a serious chill ensued a hot draught was administered and a mustard plaster was applied to the front of the chest in the hope of preventing bronchial congestion. If, despite these measures, a congestion resulted the patient was kept in his room for a day or two until the resulting fever had abated somewhat, when the invalid was again taken into the open air.

In most cases the beneficial effect of life in the open air is soon observed in decrease of body temperature and sweating, improvement of appetite and diminution in the frequency and severity of the cough. Expectoration is apt to decrease somewhat or change in character, and sleep is sounder and more refreshing. The patient usually experiences a sense of improvement which, if it cannot be put in words, is yet real to the sufferer.

Weather-limitations to the carrying out of the open-air treatment are far fewer than might be supposed. In fact, neither intense cold of Winter nor heat of Summer, neither rain, wind nor snow need be a hindrance to its adoption. Saranac patients have been known to sleep out of doors on the porches when the thermometer registered 18° below zero F. Dettweiler told me that neither wind, rain nor snow kept his patients in. It is altogether a question of protection and individual vigor.

It would be most unwise to insist that a consumptive in an advanced stage remain out in the severe cold of Winter on a cloudy windy day when his anæmia and skin-sensitiveness made it impossible to keep warm. As regards this sensitiveness to cold seen in some cases Dettweiler remarked that in some instances it became so pronounced and intractable as to constitute what might be termed a disease of the skin.

With exception of such cases, however, it is possible to keep tuberculous subjects, particularly those in an early stage, outdoors in Winter or on sunless, windy days in other seasons, provided he have suitable covering. Nevertheless, the physician must use judgment and caution in the commencement of this plan of treatment. Some invalids may take the open air in full measure from the start while others may have to begin by degrees. Of course the season of year and the local weather conditions exert material influence upon the degree of care that has to be exercised. Even a fairly well-nourished and robust person may have to practice caution in the midst of Winter, whereas in the Summer time when even nights are warm a really feeble and sensitive consumptive might be able to pass the entire day and perhaps the night on a properly protected balcony without having to accustom himself to it gradually.

What has been said thus far may lead to the conclusion that it is a simple matter to carry out this open-air treatment, but experience teaches a very different lesson. In one case we may have to consider the individual's dislike to the publicity connected with lying in bed on a gallery exposed to the gaze of passers-by. In another there is a natural timidity which causes the invalid positive fear when left alone even on a porch, not to mention the shack in the yard. In still another case the consumptive is so weak and easily chilled as to put out of the question the thought of exposing him to the night air even in early Summer or beginning Fall.

There are other instances in which one has to contend with the prejudices of either patient or friends against sleeping out of doors lest harm result from exposure to the dew and chill of the night air. Notwithstanding these or other objections the patient must be made to get the maximum amount of fresh air in some manner. Halfway measures do not answer in so serious a fight as that against pulmonary tuberculosis. It is the consideration of the many obstacles that may arise which makes me look with favor upon an ingenious arrangement devised by Dr. S. A. Knopf and described by him in the New York Medical Journal of March 4, 1905.

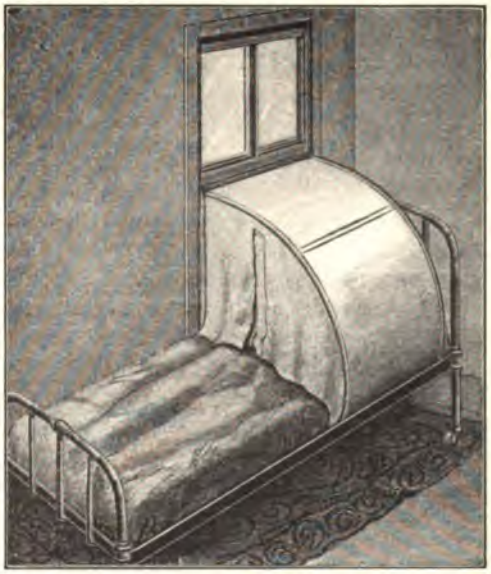


FIG. 79.—Knopf Window Tent.

This Window Tent as Knopf calls it is seen in Fig. 79 and meets many if not all the objections that can be raised to spending the night outside one's room. It possesses other advantages that will appeal to the doctor who is trying to secure a supply of pure air to the dweller in the city or to the consumptive far advanced in this complaint.

As shown in the cut the window tent consists of canvas stretched upon a framework of iron of such size and shape as to fit into the lower half of a window sash and projecting into the room, to rest on the bed and inclose the invalid's head and shoulders. The distance to which this tent must extend in a curve or quarter of an arc inward and downward must be about 40 inches or far enough to reach across a single bed. Its width corresponds, of course, to the length of the window sill.

To guard against the possible inpour of rain or snow and to shield from sun the upper portion of the tent should project for a foot or more outside the window and curve downward like an ordinary canvas awning. This latter portion could be made to work up and down on a hinge if desired and expense be no consideration. The part of the framework inside must be covered by canvas enough wider and longer than the frame to admit of its being tucked in snugly under the mattress on two sides and beneath the bedclothes on the third. By having the side of the bed placed close up against the sill of the window the patient is thus confined as far as his head is concerned in a small tent having one side freely open to the fresh air outside his room. By means of a flap in the top or at one side of the tent he can be handed whatever he needs from time to time. Whenever it is necessary to bathe or change his garments the tent can be removed altogether and the window closed for a time. Moreover, this canvas arrangement does not interfere with other possible occupants of the room or prevent conversation being held with the invalid.

Any possible objection to this window tent that might arise from the thought that a current of foul air might pass upward between the bed and the wall of the room could be met, I think, by having a strip of canvas tacked to the upper surface of the sill and stretched across the space between the window ledge and bed under which it may be securely tucked. This strip might be fastened by small flaps to the lower edge of the side walls of the tent, thus rendering the inclosure still more complete.

The circulation of air within this tent, as pointed out by Knopf, takes place in such a manner that the impure warm air rises to the top and passes out while the cooler pure air enters at the bottom and thus reaches the lungs of the invalid. If the patient endeavors to lie with his face turned toward the window his supply of fresh air is still further assured.

The expense of such a window tent cannot be great even when constructed of iron. Yet, for any case in which even this cost might seem too much I would suggest that a rough yet fairly satisfactory imitation might be constructed out of barrel hoops and unbleached cotton sheeting rendered impervious to the foul air of the room by a coating of varnish.

Sleeping Bags.—If the open-air management of pulmonary tuberculosis is to be carried out efficiently the invalid must sleep out of doors even when the nights are wintry. This should be insisted upon in the case of every fairly robust person, and only when the thermometer drops to several degrees below zero should such an individual be permitted to substitute a window tent for his shack or porch. Even 20° below zero F. need not deter a determined invalid from sleeping in a King's shack if he be provided with suitable covering.

As is well known, a person may keep warm if he be snugly inclosed by his bedclothes, that is, if he tucks his blankets under his feet and legs and pulls them close about his trunk and neck. Whereas, if he allows his covering to be spread out smoothly to the very edges of a broad bed he may feel cold no matter how thick and warm the bedclothes. A feather bed is warm largely because it permits the body to sink down into it and to be compactly surrounded by the warmth-retaining feathers.

On this principle, accordingly, sleeping bags are devised into which the patients crawl and which are then buttoned snugly about the shoulders and neck. Such a bag may be of fur, feathers or thick quilted comforters, the exterior being covered by some waterproof material. It is not necessary, however, that a sleeping bag cost more than a few dollars if made of several layers of blankets or quilts sewed together and having a thickness of newspaper between every two layers of the cloth material.

In every instance the bag should be lined with some woolen fabric and it should be long enough and wide enough to admit of some moving about by the patient when inside. Of course such a bag necessitates the confinement of the arms. This might be irksome to a few persons, but if the patient is to keep warm on a cold Winter night he cannot have his arms and hands uncovered. Although having never seen such a bag, I would suggest that one might be constructed having small extensions for the reception of the arms so as to obviate the necessity of keeping the arms at one's side during an entire night.

Head coverings are also indispensable on a severe Winter night or when a cold wind blusters about the head. All that is required for such a protection is a knit woolen cap or bag that can be drawn down so as to be tucked in around the neck leaving the face exposed. Or the openings might be three in number so as to uncover only the eyes, nostrils and mouth. The material should be soft so as not to rustle whenever the head is moved.

Warming pans or hot-water bottles may be used still further to insure sufficient warmth during the hours of sleep. If the means and environment admit of an electric warming pan this will be found the most satisfactory. One or more of the small Japanese heaters which ladies sometimes carry inside their muffs could also be placed in the bed or bag with entire safety.

Woolen night clothes should be worn by the persons who are to be exposed to the rigors of a Winter night. There is a double purpose to be met by such garments. Not only must they conserve body warmth during sleep, but they must provide protection when the person is passing from his dressing room to his berth out of doors and the reverse.

The use of this term "berth" suggests that if the consumptive reside in the country or in a village he can secure excellent quarters for open-air treatment by utilizing a woodshed at the rear of the house or by having built especially a small cheap extension or lean-to which extension or woodshed should have one entire side open to the air. Inside this shelter and nailed up against the wall could be a wooden bunk or berth which would afford a comfortable accommodation for the night. From such a shed it would be an easy matter for the patient to escape to the warm kitchen in the morning.

In concluding what I have to say concerning the manner in which this open-air treatment is to be secured I wish to utter an emphatic *protest against the idea that a room is just as good as a shack or gallery provided the windows are kept open.* I have been asked this question often by both physicians and laymen and my invariable answer is in the negative. There are several objections to a room. Open windows do not furnish sufficient ventilation unless they be on three sides and be kept wide open. In such an event they are likely to occasion a draught across the bed and a draught in a room is a totally different matter from exposure to an enveloping atmosphere under the free sky. Finally if there are windows that can be closed it is a temptation to close them on a cold or stormy night which it is practically impossible for the solicitous mother or nurse to resist.

Of course a very sick consumptive in the last stages should not be subjected to this open-air treatment in all its rigor. For such a person a large sunny south room with one or more windows on two sides must suffice. Although confinement in such an apartment is not equal to a properly built shack, still it is infinitely better than confinement in a hospital ward, a close tent or a room the windows of which are kept shut and the ventilation of which depends upon an open grate fire. Half a loaf is better than no loaf! When, in any case, therefore, this plan of management cannot be carried out in its entirety, then be content with the next best thing, but do not be content with a room in any case when it is possible by determination and ingenuity to secure complete life in the open air.

The Enforcement of Physical Rest.—It was stated under *Rules for the Enforcement of the Open-air Treatment* that rest is to be insisted upon as long as the temperature remains above 99° F. By this is meant that febrile patients do not get on well if allowed to exercise in the open air even though the body temperature average no higher than is the rule in incipient tuberculosis. By reference to preceding pages it will be seen that the average daily range of temperature in early unmixed tuberculosis is between 99.5° and 100.5° F., whereas the advent of mixed infection or the stage of softening is marked by more decided fever.

It may be thought that so slight a daily rise as that of incipient disease or an apex catarrh is of no particular moment from a therapeutic standpoint and need not be taken into account in prescribing the manner in which fresh air is to be obtained. This is an error! Experience has taught conclusively the importance of considering the temperature if a patient is to derive the most good from this plan of management.

Therefore, a patient with incipient catarrh and an average increase of body-heat of only 1 *to* 2 *degrees should be put at rest in the open air* It
is not always necessary to enforce this so rigorously as not to permi
ing in the morning, walking to the toilet room, going to the dinin
and light amusements, as fancy work, card playing, etc. But if t
disproportionate wasting and loss of strength or if the cough be un
frequent and annoying, physical repose would better be rigidly e
The rapidity with which the temperature comes down to normal a
improvement in other symptoms will usually prove surprising.

Should the febrile reaction be that of the second stage the patient must be placed *at absolute rest in bed the same as would a typhoid-fever patient.* There can be no "ifs" or "ands" about it and a nurse must be secured who will see to the literal enforcement of this injunction. I do not believe such a patient should be allowed to leave his bed for any purpose whatever, not even to go to the closet.

The same rule applies to a consumptive in the third stage who is conforming to the open-air plan of management. The higher the afternoon elevation of temperature, the more pronounced the sweating, the greater the emaciation and decline in strength, then the more vital is rigid insistence on absolute rest in the open air.

In some sanatoria there is stricter enforcement of this rule than in others, but I am a firm adherent of this policy. If there are to be any exceptions these must be rare indeed. Certainly I can conceive of no reasons that can justify an exception in any case which shows the indications stated and which has not been trying this kind of treatment already. *A consumptive patient with hectic fever should under no circumstances be permitted to make daily or even occasional visits to a doctor's office for the sake of inhalations or any other vaunted treatment.* Such a patient must be kept at complete physical and mental repose in the open air if he is to be given a chance of improvement. This rule cannot be too strongly or too often repeated, for this lesson seems as hard for the practitioner to learn as for the patient.

It is truly surprising how soon the consumptive learns to enjoy his repose in the pure cool air outside his house and how soon his symptoms show amelioration. In particular it is often found that his distaste for food gives place to a relish for the same and that his night's repose is sound, without his exhausting perspirations.

The *length of time* during which rest is *to be insisted upon* is to be determined by the degree of improvement resulting. When at length, after few or many weeks as the case may be, the temperature is found to remain *continuously at* 99° F. *or under* for a period of ten days or two weeks, then and not until then may the enforcement of rest become less rigid. Let it be said right here, however, that it is not enough for the temperature in the morning to be 99° F. or less while that of the late afternoon rises to above this point. The temperature must be persistently at or below 99° F. as indicated by the thermometer five times each day, namely, at nine, twelve, three, six and nine o'clock at night. It is on this account that I believe fully, as do some others, that if an attendant is not at hand to record the temperature the patient should do so himself, provided, of course, he is honest and has no desire to deceive. No mere verbal statement as to the temperature should be accepted, but each reading should be written down for the inspection of the physician at each visit. Some may object to this on the ground of its leading to injurious study of one's case, but I believe the benefits in the way of the doctor's control of his patient offset any disadvantage that may accrue.

One of the great aims of this rest in the open air is the speedy return of temperature to the normal. Hence any influences that may serve to maintain it must be banished. These are various, and in some persons may be such apparently trivial causes as an exciting conversation or book, the writing of a letter, a game of cards, and even the use of the hands in embroidery or other kinds of fancy work. Occasionally one meets with persons who so fret under this restraint and are so nervous that they do not get on well. Their fever keeps up in spite of the rest. In such cases much judgment is required and the physician may find a certain amount of exercise or recreation actually beneficial. Consequently, the physician's duty requires him to ascertain by trial what effect on the temperature is exerted by such seemingly trivial matters and to interdict them if the effect be found injurious.

When at length all conditions for a trial of *exercise* are favorable this is to be permitted with extreme and even, as it may seem to some, ridiculous caution. In particular its effect on the temperature is to be noted. To this end the temperature must be accurately recorded *before* any exercise is attempted. Then a short leisurely walk of five to ten minutes may be taken after which the temperature is again carefully recorded. If this be found to have risen 1 or more degrees it is an indication that the time for abandoning rest in the air has not yet come.

Accordingly, the invalid is kept at rest for another week or so after which the same experiment is repeated. When at length this short stroll is found not to be succeeded by elevation of temperature of 1° F. or more another somewhat longer walk is permitted on the next day but *never* on the same day. In this manner the effect of exercise is carefully watched until the time is ascertained when the patient can be trusted to look out for himself.

Strict Medical Supervision.—The remarks just made for guidance in the important matter of permitting resumption of exercise should emphasize the necessity for strict supervision of the patient by him who would be a successful phthisiotherapist. There are many physicians at sanatoria and health resorts who are experts in the management of tuberculous patients. I enjoy the acquaintance and friendship of many such. In every instance they exert a rigid control over the patients committed to their care, and the reason they merit the reputation of being experts in phthisiotherapy is not alone in their experience but in the fact of their never losing sight of their patients' habits and manner of daily life.

The necessity for a carefully planned and regulated daily regimen on the part of the tuberculous is far greater than is generally recognized and cannot be too strongly expressed. The tuberculous individual generally shows a lack of persistence in his obedience to orders that springs, possibly from his hopefulness or is a manifestation of that mental peculiarity
mention was made under Symptoms. Even a conscientious con
will every once in a while omit some detail or commit some in
without seeming conscious of what he is doing.

Therefore, if a tuberculous invalid is to be kept up to the
should be given written or printed instructions that cover practic
hour of his waking day. It is so easy for a person of this type to

in his slow climb upward to health that if his progress is to be uninterrupted he must live in accordance with a definite regimen, and the medical attendant must equally faithfully inform himself at each visit as to how this system of daily life is being followed.

The tuberculous is not expected to be a slave to the dictates of a tyrannical master, but if he has confidence in his medical adviser he should be scrupulous in his obedience to his injunctions. If he has not confidence in his wisdom and skill then he would better discharge him for another.

Exercise.—It is especially in the matter of exercise that there is danger of the tuberculous doing himself harm after having been given liberty to exercise. If a convalescent will be a fool by carrying his exercise to the length of exhaustion, by neglecting to carry wraps or rubbers or umbrella when weather is threatening, by going insufficiently protected against rain or snow when it is storming, by dissipating with wine, women and song, by committing the thousand and one indiscreet acts to which the light-minded are prone, then of course no amount of advice and sound admonition from his doctor will avail to save him from the consequences of his acts.

The kind of exercise permissible for the convalescent must be determined by the extent and nature of his pulmonary changes, by the vigor of his regained health, the presence of complications, etc. One who has recovered from an apex catarrh can probably return slowly to field sports, extensive pedestrian excursions, hunting, fishing, rowing, riding, etc. He may with benefit practice some system of breathing for the expansion of his chest.

The convalescent consumptive, on the other hand, who has quiescent cavities in his lungs or extensive fibrosis must attempt these things with extreme caution and would better leave the more severe of them untried.

Hydrotherapy.—Another therapeutic measure about the value of which considerable diversity of opinion prevails is the use of water. It appears to have been introduced by Brehmer and Dettweiler and to enjoy greater popularity in German sanatoria than in the United States. The object of hydrotherapy in this class of patients is to stimulate circulation in the skin and render it less sensitive to sudden changes of temperature. It is found, in addition, to invigorate the circulation generally and hence to promote the removal of tissue-waste and to improve appetite and digestion.

The *method of its application* varies greatly and depends largely upon the strength and reaction of the individual. Persons with an incipient apex catarrh that has not yet led to much debility may begin with a tepid sponge bath in the morning either taken by themselves or given by an attendant according to their strength. In every instance the patient must be enjoined to take his sponge so rapidly as not to let the feeling of warmth marking the reaction be succeeded by a chill.

Those who are less vigorous would better be bathed by a nurse who should bathe one part at a time and beneath the bedclothes, if considerable sensitiveness to cold exists. First the chest is rapidly gone over with a cloth wrung out in tepid water and is then rubbed dry before the arms, trunk and lower extremities are treated in the same manner.

Patients who are much reduced in vitality are first to receive a dry rub and then, as strength is gained, to be sponged in the same careful manner as above with alcohol, then with alcohol and water and afterwards with plain water. Care must be exercised not to fatigue weak patients with these morning rubs, and not to come to the use of water alone until the ability to establish a good reaction has been demonstrated. Judgment as well as skill is required to give this treatment successfully and hence it should not be left to ignorant or inexperienced attendants to determine when the more vigorous forms of hydrotherapy are to be employed.

As the previously weak and sensitive increase in resistance they may be given a sheet-rub or a sponge bath as circumstances render advisable, it being remembered always that the promptitude and degree of reaction are indications of the invalid's ability to endure the baths.

By degrees the vigor of the treatment may be increased until at length an individual becomes capable of withstanding and even relishing a *douching or shower bath.* This is a very stimulating form of hydrotherapy which demands much caution in its application. The douche should begin with warm water which may be gradually reduced in temperature and it should not exceed 5 to 10 seconds in duration, the shower of drops being quickly passed over the body from neck to heels. Immediately thereafter a dry sheet is to be cast about the body and the drying process commenced by the attendant.

In the course of time the patient may be able to shower himself, but only very vigorous persons who have become inured to this heroic treatment are to be allowed this privilege.

In suitable cases a simple and highly stimulating bath may be given by the dashing over the body of a pailful or two of cold water, this being instantly followed by a vigorous rub during the time of reaction. I have known a man who had regained his health in Colorado to take a daily morning plunge in a cold mountain stream and to declare it made him "feel just fine."

Any form of shower bath or a plunge into a tub of cold water is a very efficient but at the same time dangerous means of applying hydrotherapy and is not to be allowed by the physician or undertaken by the convalescent if health and strength have *not been nearly or quite regained,* or if the individual is *subject to hæmoptysis.* Unfortunate results have been reported in a number of instances (Walters), and hence such energetic hydrotherapy is now less employed than formerly.

A hot tub-bath should never be permitted, in my opinion, as I believe it to be debilitating. A weekly or semiweekly bath with warm water (93° to 98° F.) and soap may be allowed, however, for the sake of cleanliness, but orders must be given against remaining any longer in the tub than is necessary. It is often well to follow such a bath with a quick shower of cold or nearly cold water. A temperature of 78° to 80° F. is cold en for such douches in ordinarily robust convalescents.

Dietetic Management.—It must not be supposed because the consid tion of the nutrition of the tuberculous has been reserved until now th is less important than the measures previously discussed. It is a matter that must receive the physician's attention at the outset of treatment and must be intelligently supervised throughout. There is no other one thing that is likely to occasion as much perplexity and annoyance, and partly on this account it is the rock on which the bark splits.

Now and then a tuberculous individual is encountered even in an advanced stage whose appetite and digestion appear unimpaired, but in the overwhelming majority of cases, even in the incipient stage, the patient either has a positive repugnance to food or is found to eat but sparingly of articles as fruit and cereals which lack sufficient nutritive value. We generally find also that when a consumptive is able to eat or is encouraged to eat by physician or nurse no account is taken of the height of his fever or of the appropriateness of his fare. Accordingly, an attempt will be made in these pages to consider these various phases of the problem confronting us in the nourishment of tuberculous patients.

As remarked by Fraenkel, the three measures, namely, life in the open air, exercise and hydrotherapy, all tend to increase metabolism, and hence the aim of dietetics in this disease is to replace tissue-waste and build up new tissue, that is, to increase body-weight. Cases are seen now and then in which the disease seems to make progress in the lungs despite gain in flesh, but ordinarily the increase in weight may be regarded as a fair criterion of the gain over the disease. Accordingly, the patient should be weighed each week for the purpose of ascertaining if he be consuming sufficient food or if this be of the right kind.

From the standpoint of physiology, however, any system of dietetics cannot be considered scientific which is based on body-weight alone and does not take into consideration the number of calories contained in the food.

According to Voit and Rubner a healthy grown man requires for his daily nourishment 120 gms. albumin, 50 of fat, and 500 of carbohydrates; or, expressed in calories, 492 of proteids, 465 of fats and 2,050 of carbohydrates. Estimated according to body-weight and whether the individual be at rest or at light work, we have the following: at rest 30 to 35 calories for every kilogram of body-weight, at light work 40 calories per kilogram. As a kilogram may be said to equal about 2¼ English pounds, an adult at rest requires about 15 calories for every pound of body-weight, and at light labor about 18 calories per pound. On this basis, therefore, a man of 150 pounds weight would need when at rest about 2,250 calories and when at light work about 2,700 calories. Chittenden's investigations indicate that Voit's figures are very much too high for normal individuals. Especially that the average amount of proteids consumed is excessive.

The tendency of pulmonary tuberculosis is to progressive loss of weight, and hence its popular name consumption. Consequently, if we are to arrest this tendency to emaciation and at the same time to improve the quality of the blood and build up tissue round about the tuberculous foci, we must order a dietary containing more calories than would be required by a healthy grown man under similar circumstances.

The exact number of calories required for each individual cannot here be stated. It must be determined by the weekly gain in weight, the patient's ability to digest and appropriate without digestive or other derangements, the stage of the disease and other factors. It is often an error to force the feeding after symptoms have become quiescent in the same manner as when emaciation was progressing. After the individual has regained or surpassed his normal weight it is well to relax somewhat in the insistence on superalimentation and to restrict the patient now and then for a brief period to fasting or to an exclusive milk diet for twenty-four hours. In this way serious attacks of acute indigestion may be avoided.

Chittenden's experiments and conclusions appear to be influencing some physicians to the belief that we often make the mistake of overfeeding our tuberculous patients. While fully convinced of the likelihood of harm by a too long continued or too rigid hyperalimentation in every case, I yet cannot believe that so great a decrease in proteid diet as is advocated by Chittenden can be well for patients who suffer from a wasting malady.

Neither am I in accord with those who would eliminate all flesh foods from the dietary of the tuberculous. I am satisfied that in many instances the amount of meat and other forms of animal food consumed is entirely too large and I am willing to concede that now and then persons are found who thrive on a purely vegetarian diet. Yet it is my conviction that this class of invalids is most likely to gain in strength and weight if they consume a due proportion of their proteids in the form of flesh, fish, fowl and eggs.

The annexed table of food stuffs has been prepared from the list given by Cornet and from Atwater's Chemical Composition of American Food Materials in the hope of its proving of aid to the reader in prescribing the dietary of his tuberculous and other patients. Atwater's list comprises an enormous number of food materials, and in the preparation of this list only those articles have been selected which are of common use. Moreover, only those figures have been taken which express averages. It will be observed that Cornet's figures are per 100 grammes of weight, whereas Atwater's are per pound. It may be stated also that the articles mentioned are all as cooked.

FOOD MATERIALS.	CORNET. Per 100 grammes.	ATWATER. Per pound.
Animal Food.		
Beef, raw	119	
" cooked	209	
" roasted	214	
" smoked	255	[illegible]
" steak, loin		[illegible]
" corned		[illegible]
" dried		[illegible]
Veal, cutlet, raw	142	[illegible]
" roast	230	[illegible]
Mutton, leg, roast		[illegible]
Lamb, " "		[illegible]
" chops		[illegible]
Pork, fat	313	[illegible]

FOOD MATERIALS.—*Continued.*	CORNET. Per 100 grammes.	ATWATER. Per pound.
Animal Food.—Continued.		
Bacon	617–761	...
Calf brains, raw	140	...
Sweetbreads	90	775
Kidney, stewed	...	600
Tongue	393	1,455
Fowl, breast	100	...
Breast of goose	381	...
Ham	438	...
Cervelas sausage	445	...
Liver "	290	...
Fish.		
Pike	72	...
Trout	106	...
Salmon	133	660
" smoked	224	...
Codfish	61	...
Perch	76	...
Pompano	...	375
Red Snapper	...	225
Shad	...	380
" roe	...	600
Spanish mackerel	...	525
Whitefish	...	325
Bluefish	...	670
Herring, pickled	246	...
Sprats, smoked	243	...
Shell Fish.		
Oysters	20	235
Clams	...	340
Lobster	...	390
Caviar	278	...
Vegetables.		
Potatoes	127	505
" chips	...	440
Asparagus	18	220
String beans	41	95
Navy beans	193	...
Peas	75	...
" green	...	540
Carrots	41	1,700
Spinach	166	260
Onions, cooked	...	190
Corn, green	...	455
Tomatoes	...	105
Beets	...	185
Vegetable Food.		
Bread, brown	...	1,050
" rye and wheat	203–232	1,190
" white	229–260	...
" " home made	...	1,225
" toasted	...	1,420
Pumpernickel	229	...
Zwieback	232–358	1,970
Crackers	374	...
Cookies, molasses	...	1,910
" miscellaneous	...	1,875

Food Materials.—*Continued.*	Cornet. Per 100 grammes.	Atwater. Per pound.
Vegetable Food.—Continued.		
Doughnuts		2,000
Pie, apple		1,270
" custard		830
" mince		1,190
Pudding, Indian meal		815
Rice		525
Dairy Products.		
Milk	67	
" skimmed	40	
Cream	215	
Butter	756–807	
Buttermilk	41	
American cheese	179	
Swiss cheese	340	
Eggs.		
Scrambled	188	
One egg, raw	70–80	
Omelet	237	
Fruits.		
Apples		290
Bananas		460
Blackberries		270
Cherries		365
Grapes		450
Muskmelon		185
Oranges		240
Pears		295
Pineapple		200
Prunes		370
Strawberries		180
Watermelon		140
Apricots		340
Peaches		220
Nuts.		
Almonds		3,030
Brazil nuts		3,265
Butternuts		3,165
Chestnuts		1,125
Cocoanut		3,125
Hickory nuts		3,345
Peanuts		2,560
Walnuts		3,285
Pecans		3,455
Miscellaneous.		
Chocolate		2,860
Cocoa		2,235
Cereal coffee		
Bean soup		
Chicken soup		
Clam chowder		
Koumiss		
Beef juice		
Cane sugar	406	
Macaroni	353	
Whisky	280	

The *dietary* of the tuberculous must be based on other considerations than that alone of supplying the requisite number of calories. It is one thing to tell a patient to consume a certain amount of certain kinds of food and quite another to get him to do it. Furthermore, the physician should appreciate the value and necessity of the three great constituents of nourishment, proteids, carbohydrates and fats. In the minds of many it is albumin or the nitrogenous portion of the dietary that is especially needed by consumptives, and hence they feed their patients almost exclusively on meat and eggs or, as some have advocated, on finely divided lightly broiled almost raw beef, washed down with copious draughts of hot water.

Whereas such a diet may be well borne by tuberculous patients having a hyperchlorhydria it is not the kind of nourishment indicated for the majority. Experience teaches that a generous mixed diet meets the requirements of the largest number. There are some who, convinced by the experiments and teachings of certain investigators, advocate a purely vegetarian diet for this class of invalids. I deny not the richness of such a dietary in proteids, nor do I deny its suitability to some cases of constipation or putrefactive intestinal fermentation, but a bulky vegetarian regimen cannot be endured by all.

Therefore, I stand by the experience of the majority of phthisiotherapists which teaches that the diet most suitable to the needs of the tuberculous is one composed of a generous proportion of animal proteids together with more or less fat and carbohydrates according to the degree of emaciation and the digestive power of each individual. In particular I would emphasize the necessity of *fats* in most cases in forms that are not repugnant and are readily assimilated. These are especially butter and cream, though the fat of bacon or pork is excellent when relished and not distasteful. A moderate layer of fat (panniculus adiposus) is not only conducive to beauty of appearance but serves as a storehouse of energy on which to call whenever, for any reason, unusual demands are being made on the system, and hence it should render one apprehensive if a tuberculous individual fails to take on fat.

Vegetables are very wholesome for many reasons. The leguminous ones supply a relatively large percentage of proteids, and most of them contain carbohydrates and inorganic salts as phosphates which are very serviceable. Farinaceous articles made from grains, particularly from wheat and corn, are convertible into fat, at a considerable loss to be sure, but are of positive value in this and other ways. Sugars are of especial aid in the formation of adipose tissue, while *fruits*, in addition to supplying this carbohydrate serve as a relish and favorably affect intestinal action.

Milk is indispensable and far more nutritious than most persons appreciate. A glassful (8 ounces) of rich milk is said to possess a caloric value equivalent to 2 raw eggs, or 2½ ounces of cooked beef or 2¼ ounces of white bread. Eggs are also exceedingly valuable articles of food because highly nutritious and in a raw state most digestible. As we shall see, therefore, we possess in these two articles of food agents for the building up of tuberculous patients without which we would many times be at a loss how to proceed in this most difficult problem.

Rules for feeding this class of invalids may be formulated and hence seem very simple, but he who would successfully overcome the difficulties of this part of management must have derived his knowledge from actual experience with consumptives or from personal experience with a delicate stomach. There is a saying to the effect that appetite grows with feeding, and this is assuredly true of most tuberculous patients. They can be made to take and digest a hearty meal if influenced in the right manner.

Suggestion on the part of the physician, or *moral suasion* as it is called, is often a potent factor. I think I have never yet seen a tuberculous patient whom I could not convince of his ability to swallow a raw egg. Of course the suggestion must be backed up by having the egg properly dressed. So it is with the eating of a meal. Make the consumptive believe in his ability to eat and digest the food set before him, but as Cornet says, do not commit the error of setting so much before him that he could not devour it all were he a hungry healthy man.

Five times a day are often enough for most patients to take nourishment. Unless some special consideration exist for more frequent and light supplies of nourishment let there be the ordinary three substantial meals, breakfast, dinner and supper; to which may be added a light luncheon in the middle of the forenoon and another in the middle of the afternoon. This number can usually be tolerated by persons with incipient or early disease whose power of digestion has not been reduced to a minimum by a lifetime of dyspepsia or foolish disregard of their requirements in this direction.

Persons who are very feeble may do better if given light nourishment at shorter intervals, that is, a little every three or even two hours from the time they wake in the early morning until they go to sleep at night. Such advanced consumptives may require a cupful of milk or soup when they wake in the night drenched with perspiration and cold. Under such circumstances the drink should be hot and may contain a half ounce or less of brandy with advantage.

Clock-like precision and regularity in the time of serving nourishment is considered of utmost importance if one is to cultivate in tuberculous persons the habit of taking food and a relish for the same. The indiscriminate munching of titbits must never be permitted and the nurse must be made to realize that when the hour for a meal or luncheon has arrived it must be on hand. A sickly person who feels hungry at the proper time will often grow so faint when kept waiting that the food goes against him when at last it does arrive. Therefore, the physician should write down the exact hours for nourishment and must see that his directions are obey

The food must be *well prepared and attractively served.* Scarce one rule is more important than this. Not only is good cooking e to good digestion but it renders food savory, and unappetizing via better thrown away than offered to a consumptive with a delicate s The odor of food has much to do with its attractiveness and hen the patient's appetite. It must appeal to the eye also, since repug excited by dirty linen or a mass of uninviting food dumped on to a plate in a promiscuous heap, scarce suitable for a railroad laborer. Such are by no means unimportant details.

It is not advisable for patients to take their meals alone, since by so doing they are apt to eat in a hurry, masticating imperfectly and often slighting certain dishes. An individual who can control his cough or who is in a tolerably good condition may be allowed to join the family provided the effort does not raise his temperature. It is partly on account of the beneficial effect of cheerful company that the inmates of closed sanatoria are allowed to eat together as long as their physical condition does not render this unwise.

To overcome anorexia is in many instances the special task of the physician, particularly in the case of consumptives in an advanced stage. The sufferer is either unable to eat or fancies he is, or having some desire for nourishment he finds his appetite gone after having taken a few mouthfuls. As a rule, this inability to take food does not depend upon actual disease of the stomach but is the result of the infection and is joined to a lack of will power, in consequence of which the patient cannot force himself to eat. Such persons are extremely notional, as it seems, and try one's patience to the utmost.

If the symptoms of infection be pronounced it is sometimes sufficient to put the consumptive at rest in the open air when, after a few days, his appetite will evince signs of returning. Then as nourishment is taken at regular periods and as the physician uses suggestion, both the ability to take food and the tolerance of it are found to increase. It will be found in these cases likewise that the morning bath favors the development of appetite, while the toilet of the mouth is very essential.

When such simple measures do not suffice, recourse must be had to other procedures in the hope of stimulating a healthy hunger. Cornet mentions a Priessnitz bandage on the epigastrium or the use of faradism, citing an instance in which, after the complete failure of other means, a half hour's application of the faradic current was followed by the ability to consume and digest a substantial meal. He mentions also the beneficial effect of hydropathic measures in general.

It is not a good plan to resort to bitter tonics for improvement of appetite, inasmuch as the sufferers have usually been taking all sorts of remedies and the anorexia is sometimes the result of drug-taking. I prefer to try the effect of a glassful of hot water half an hour before breakfast, the juice of an orange, or 6 ounces of milk warmed to an agreeable degree but not boiled. If to the milk is added one-third as much Seltzer, Apollinaris, charged Vichy, or any other effervescing alkaline water the effect on the stomach is usually excellent. Strange to say, such a drink on waking does not interfere with the taking of breakfast a short hour subsequently and acts as a gentle stimulant to the consumptive who has slept poorly and wakes feeling damp and generally uncomfortable. In cases of marked feebleness a little rum or cognac may be put into the milk with advantage.

Another plan which I have found highly efficacious in restoring appetite is to confine the patient to an absolute milk diet for two or three days. A glassful of warm, not hot, milk is administered every three hours and absolutely nothing else; in particular all medicines are stopped and the invalid at the same time is put to bed in the open air.

One instance stands out in my remembrance which responded to this simple plan after two days. At the end of that time the girl who was burning up with hectic fever and had declared she could eat nothing asseverated she was starved and must have something to eat. As a matter of fact she *was* starving, inasmuch as the three pints of milk she had consumed daily did not contain the requisite number of calories to sustain her, although in a state of rest. Accordingly, it will not answer to continue such an absolute milk diet indefinitely, and if, after three or four days this fails to restore appetite it must be abandoned or reënforced by other foods. To this I shall return anon. Very reduced consumptives in an advanced stage cannot always be made to take much nourishment no matter what means to this end are tried. In fact, it is hardly worth while to torment them since they are too far gone to be saved anyhow. It is better to give them as much nourishment as they can be induced to take in a simple easily digested form and in small quantities at intervals of two hours.

A young married woman who was slowly dying of acute pneumonic phthisis got into such a state that she vomited everything she took. Her stomach was given an entire rest for a number of hours after which her sister who was acting as nurse began feeding her with teaspoonful doses of milk and limewater every five minutes with the result that the nausea and vomiting gradually ceased. Slowly the intervals were lengthened and the amounts administered were augmented until at length the stomach regained its ability to accept and digest more substantial nourishment.

"*What may the patient have to eat?*" This is the question invariably asked of the physician who sees a consumptive for the first time either alone or in consultation. On this account it seems well to state the hours at which nourishment should be taken and, so far as practicable, to outline a menu suitable for this class of patients.

For persons whose condition does not contraindicate solid food: Breakfast, 7 to 7.30 A. M.; 8 ounces of milk with an equal amount of tea, coffee or cocoa if desired, but if not, then another glassful of rich milk reënforced by the addition of cream; cereal with cream and sugar; fresh salted or smoked fish or bacon and one or two soft-boiled eggs; beefsteak, lamb chop or sweetbread broiled, or a well-seasoned hash of roast or corned beef; or an omelet with minced ham, potatoes, if desired, or some relish as radishes, cucumbers, or fresh sliced tomatoes dressed with oil and vinegar; buttered toast or waffles; fruit, fresh or canned, with cream, this latter at th
rather than at the beginning of the meal.

Luncheon at 9.30 or 10 A. M., according to the heartiness of the
fast; 8 ounces of rich milk with a raw egg, or a slice of buttered brea
thin meat sandwich, or bread with marmalade.

Dinner at 1 to 1.30 P. M.; substantial soup or raw oysters or cla
thick layer of caviar on toast; fish and potato; roast meat or fowl

with several kinds of vegetables or *macaroni au gratin*, bread and butter; a salad with crackers and cream cheese, a dessert of pudding, custard, ice cream, jelly and cream, or canned or preserved fruit with cream; one to two glassfuls of rich milk.

Afternoon luncheon at 4.30 to 5 P. M.; a glassful of milk reënforced with cream or, if dinner was very hearty, a cupful of tea with plenty of cream and sugar; light biscuits or delicate cakes (cookies), or a cupful of broth, or beef tea if there is danger of the invalid's growing tired of the milk.

Supper at 7 P. M.; some meat or fowl either cold, freshly cooked or hashed; oysters in any style; a vegetable or fruit salad with cream cheese or cottage cheese; bread and butter; cooked or canned fruit with cream; one to two glassfuls of rich milk.

Another light luncheon may be added at 9 to 10 P. M., if supper was not hearty or there is a desire for more nourishment to carry the patient through the night. Should the consumptive waken after midnight and cough or be in a perspiration it may be well to administer a cupful of hot milk with liquor or hot beef tea which often promotes sleep and cessation of cough.

Raw eggs are an article of nourishment of immense value to tuberculous patients and should be taken in addition to all other foods. They may be taken very advantageously at the close of the principal meals, "on top of everything else," as I am in the habit of saying. Persons are almost sure to make a wry face at the mention of raw eggs and to assert they cannot swallow them. Such an assertion I never allow to go unchallenged and I invariably reply "Yes you can; if you only make up your mind you can!" or other words to that effect. I then point out to the family or nurse the folly of letting the patient prepare the egg himself or of holding the glass, smelling of the egg and thinking about it.

The raw egg must be dropped into a small wineglass without breaking the yolk after which it is dressed in any one of several ways. It may be seasoned with salt, pepper and vinegar the same as a raw oyster, or it may be covered with lemon juice, or a little wine (sherry) or cream may be poured in over the egg; it is then carefully stirred with a spoon so as to mix cream and white together without breaking the yolk. Thus prepared the egg is brought to the patient who is told to swallow it down without stopping to look at it or to think about it.

I instruct my patients to begin with one raw egg after each meal on the first day and to increase by one egg each day thereafter until at length as many as four raw eggs are ingested after each of the three principal meals. This number is then to be continued until ordered otherwise. This number of raw eggs will furnish 840 to 960 calories and increase greatly the nutrition of the tuberculous patient.

If a consumptive is too feeble to digest solid food and is to be sustained on milk and eggs when at rest he must consume, when estimated in calories, 2 quarts of rich milk together with 20 eggs or 3 quarts of milk and 12 eggs. This can be done if they are administered at regular intervals and if the eggs are swallowed with unruptured yolks instead of having them beaten up in the milk. This latter is not so digestible a manner of giving eggs to some persons but may be adopted when the eggs are not disliked or do not disagree. Eggnog which is a raw egg beaten up with liquor and sugar does not agree with all patients and is objectionable on that account.

Although patients are now and then met with, even in the incipient stage, who display an almost incorrigible repugnance to food of the kind and amount requisite for the needs of their bodies, still it is, for the most part, consumptives with hectic fever and marked emaciation who tax our skill and patience. Therefore some additional remarks seem advisable concerning the nourishment of this class of sufferers. If one will reflect for a moment on the coated tongue and digestive disorders so common at this stage of the disease he will readily perceive how unlikely are the ingestion and proper assimilation of the necessary amount of food by the bedridden consumptive who falls into his care after months of drug therapy.

Liquid nourishment is the kind suitable to patients with a temperature range above 101.5° F. and still more when it rises in the afternoon to 103° F. or higher. In such an event, therefore, the dietary must consist of milk, broths and soups reinforced by prepared foods and fruit juices. Raw eggs are also capable of being digested by most of such invalids and, when so, should be given in such numbers as will materially swell the total calories. Of the prepared foods which are rich in proteids may be mentioned tropon, which is pure animal albumin, plasmon made from skimmed milk, roborat from vegetable albumin (Fraenkel), plantose, also claimed to be rich in vegetable albumin, somatose, and lens diet made from dried peas and convertible into a very palatable easily digested porridge. Buttermilk, koumiss, kefir, casoid and matzoon also form valuable additions to the dietary suitable for these cases. One may peptonize milk or, with an essence of pepsin, prepare the so-called junket and whey which sometimes prove very acceptable to febrile patients. Wine and lemon jellies may be likewise of service since they are of considerable nutritive value.

Wines, liquors and even beer should not be omitted in this list since, when they do not too greatly stimulate the pulse and cause flushing of the surface through vaso-paresis, they furnish positive aid in maintaining strength. Their use should be attended with great caution, however.

In chronic cases displaying normal or subnormal temperatures in the forenoon and fever in the after part of the day, solid or semisolid articles may be taken and digested during the morning hours, and in the afternoon the liquid and prepared foods mentioned above.

The foregoing dietary is not supposed to be complete but is furnished as a guide or suggestion which may help out the practitioner when the whims or idiosyncrasies of the consumptive or the inquiries of the mother have put him at his wits' end. For additional and more complete food for invalids he must consult works especially devoted to dieter

The Hygiene of the Sick-room.—Sooner or later, in most cases sumption, the phthisiotherapist is called on to minister to the con prolong the life of the sufferer without hope of restoring him to Because of the stress of weather or on account of the unfortunate ment of the patient it is impracticable to keep him in the open air must be kept in his bedchamber. For the sake of protection to the family this room must be converted into a private-hospital ward as nearly as may be compatible with the patient's comfort and peace of mind.

Carpets, draperies at the windows, rugs and articles of ornament that catch and retain dust are extremely objectionable from a sanitary standpoint and ought to be removed. When finances permit, draperies may be replaced by pretty cotton prints and the floor may be covered by khaki or canvas, or may be painted a bright color, or a cork carpeting may be laid which is less noisy. Upholstered chairs should be removed for pretty wicker furniture that may be washed from time to time. The room should be made as hygienic as possible and yet not left bare and depressing. An intelligent patient or one having the welfare of his dear ones at heart will not seriously object to such changes when their object is explained.

Sweeping and dusting in this room should *never* be permitted, but the walls and floors must be wiped with a wet cloth, the latter every day or two and the former as often as once a week.

Above all, the windows of the room must be kept open day and night. Surely enough has been said already to make plain the reasons therefor. In one case, that of a Swede boy with advanced tuberculosis and in such circumstances that change of climate was out of the question, I so impressed the mother and patient with the necessity of pure fresh air that he slept with his window wide open although the thermometer fell on several nights to 20° below zero. He did not once take cold and he became so fond of the cold pure air that he afterwards told me he could not sleep with his window shut.

The atmosphere of the consumptive's room should smell as sweet and fresh as the outside air but dangerous draughts must be shunned by use of folding screens, etc. It is in this class of cases that Knopf's window tent would be of immense value. There is no objection to artificial heat in the room provided dust and smoke are not engendered by poking of the fire in stove or grate. Doors opening into adjoining apartments should be kept closed.

Woolen blankets would best be kept covered by a sheet of cotton or linen or by a counterpane of same material which can be washed whenever soiled by sputa or otherwise. In a word, everything in the way of clothing connected with the consumptive should be kept scrupulously clean and be changed frequently. As soon as any article becomes soiled with sputa it should be put into the wash and boiled or subjected to the action of moist steam for twenty minutes. In fact the dangers of impalpable sputum are such as render the boiling or steaming of all bed and personal clothing advisable.

All utensils handled by the consumptive as knives, forks, spoons, glasses, cups, plates and even napkins should be boiled as soon as removed and not set down where by any chance they may be subsequently used by others.

The consumptive does not need to be shut off from all communication with his dear ones as if he were a lunatic or dangerous wild beast, but he must not kiss or be kissed on the mouth, must have his hands and face washed often and thoroughly, especially after a coughing fit, must not fondle young children, must protect his mouth when in the act of coughing or sneezing and, in short, must exercise all those precautions which have been insisted on as necessary for the prevention of tuberculous infection of those about him.

Collection and Destruction of the Sputum.—Of all measures connected with the management of tuberculous patients none is more important than this. Much thought and investigation have been bestowed on this matter since it is not so simple a problem as might at first seem. Persons who remain at home or in an institution should expectorate into some sort of receptacle which can be easily and thoroughly cleaned and disinfected from time to time.

Cuspidors may be of porcelain, enameled iron, tin or any other metal, but in construction should be wider at the bottom than at the top to prevent them from being easily upset and their contents spilled. The opening should be sufficiently broad, however, to make the spitting into it easy and should not be covered by a lid. Cuspidors consisting of a metal framework holding a cup or box of heavy paper or pasteboard shellaced have been recommended because the paper receptacle can be burned but the expense is prohibitive for persons of moderate means and they possess no advantages over those of an indestructible sort.

The cuspidors may be partly filled with some disinfecting solution or with plain water, but the fluid must not be so deep as to splash over the sides or upon the floor when a mass of heavy sputum falls into it. A 1-per-cent solution of corrosive sublimate may be used but is not to be recommended because of its hardening action on the albuminous constituents of the sputum and the length of time required, therefore, to destroy the bacilli. Solutions of carbolic acid and allied drugs are also recommended but, on the whole, nothing is better than simple water which may be emptied down the water-closet several times a day, after which the cuspidor should be thoroughly washed in running water. In the country or in towns where there is no sewerage the cuspidors may be filled with sawdust or some other substance which can be burned.

Sputum-cups.—Tuberculous patients who do not remain at home within easy reach of a cuspidor or who are confined to bed need some portable receptacle for their expectoration. To this end so-called sputum-cups have been devised. They are kept in stock by dealers in medical supplies and present a great variety in shape and composition as shown in the annexed figures (80–81). One of the best known is that invented by Dettweiler but, although constructed with a view to its being carried in the pocket, it is open to serious objections. The opening into which the person must spit is small and is protected by a lid. Consequently it is apt to become soiled and is not easy to clean and disinfect. Knopf has invented one which has a funnel-shaped top projecting down into the cup on the principle of the ink bottle so that in case the cup is turned over its contents will not escape. The funnel-shaped cover is removable and therefore facilitates cleaning.

There may be found in the shops two forms of sputum-cups which, being made of thick paper, can be burned and are inexpensive. One of these is in two parts, the bottom to receive the expectoration and the cover to slip over it, after the plan of a cigar case. This cup is flattened and occupies but little room in the pocket. The other pasteboard cup is also flattened and is provided with a cover or lid. It is lined with some sort of waterproof material, being in this respect good. Because of its having a lid that cannot be kept very securely closed it is not so desirable as the one previously described.

The conspicuousness of a sputum-cup is an objection to its use in the minds of many patients and on this account it is recommended that the consumptive expectorate into his handkerchief or pieces of cloth or of paper that may afterwards be burned. Cornet objected strenuously to the use of the handkerchief, but Beninde is said to have proved that danger of spreading infection from the handkerchief does not exist if it be boiled before the lapse of twelve to fourteen hours and in particular if the expectoration be so copious as to keep the handkerchief moist. Personally, I do not approve of its use for this purpose, and yet, if so employed it should be carried in a separate pocket lined with rubber cloth and it must never be thrown into the family wash before being sent to the laundry. Neither should it be allowed to become dry and remain unboiled for longer than the twelve hours recommended by Beninde.

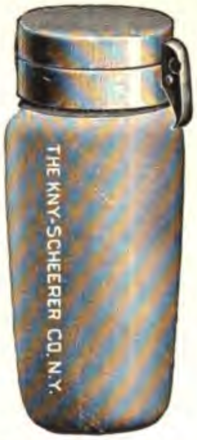


FIG. 80.—Knopf sputum-cup.

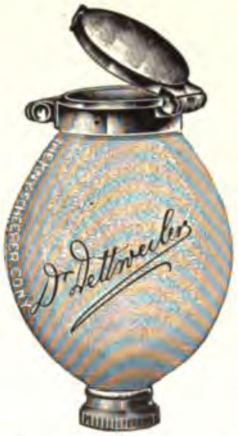


FIG. 81.—Dettweiler sputum-cup.

If very poor consumptives or bedridden patients expectorate into cloths or pieces of tough paper, these must be burned and not permitted to lie about to dry. When absent from home such pieces of cloth or paper are to be folded and placed in a suitable rubber bag or metal box until the person reaches home when they are to be burned. The lips and hands of the bedridden consumptive should be washed by the nurse after each expectoration.

Impalpable Sputum.—That bacilli may be thrown into the surrounding atmosphere by each act of coughing or sneezing, has been proven, and has been mentioned already in these pages. Consequently, every consumptive *should be taught to hold a handkerchief or other cloth in front of his mouth* whenever he coughs or sneezes and such protecting cloth should be boiled the same as if it contained visible expectoration. Indeed it would be well if every person, whether tuberculous or not, were taught to cover his mouth during the act of coughing or sneezing for nothing is more unpleasant than to have a spray of moisture cast into one's face, especially if the breath be foul.

Toilet of the mouth is closely associated with the matters of which I have just been speaking. The individual in the incipient stage should thoroughly brush his teeth and rinse his mouth after each meal as well as on rising, and should use some antiseptic solution recommended by his physician. He would do well, in my opinion, to scrape off the tongue at the same time. If his teeth be decayed they should be put in order by a dentist. Bedridden consumptives should have their teeth and mouths attended to in the same manner by their nurses.

The *use of tobacco* by tuberculous patients is not to be indorsed for a number of reasons. Smoking tends to excite cough and if indulged in to excess, injuriously affects the general health. The tuberculous who would regain health cannot afford to retain any habit that is likely to retard his recovery.

Symptomatic Treatment.—Although fully believing that no plan of management is so likely to control ordinary symptoms as the hygienic and dietetic measures already considered, still it is recognized that successful phthisiotherapy must include the occasional and intelligent use of remedial agents of another sort. Some of the symptoms calling for especial consideration will now be discussed.

Control of Cough.—This is important and is the most common subject for inquiry on the part of the consumptive. Do not prescribe a sedative, as is but too frequently done, until other measures have been exhausted and the cough robs the patient of rest. In the first place, the larynx should be carefully examined if necessary by a laryngologist. If no cause of irritation be discovered then the patient should be told plainly and decidedly that he must learn to inhibit the cough himself.

Let it be explained that coughing is harmful when it does not result in expectoration and that for the most part expectoration is necessary only in the early morning, again at midday and before retiring for the night. Between times he must resist the desire to cough, and it is truly surprising how ready, intelligent patients are to act on this suggestion and how they succeed.

Cough is especially common after the taking of food and then is o due to irritating particles adhering to the pharynx. In such instances offending material may be removed by gargling with water or some a septic solution and the cough be allayed. In some advanced cases the sy tom appears to be an expression of reflex irritation and may result in em

In these cases a sedative may be necessary but a small dose of strychnine ($\frac{1}{60}$ of a grain t. i. d.) will sometimes lessen the irritability and aid the sufferer materially in controlling his cough.

In the second and third stage the desire to keep up a frequent hacking without expectoration may be allayed by the wearing of a respirator in front of the mouth. The kind formerly much prescribed by me shown in Fig. 6 is called a Perforated Zinc Respirator. The sponge in front is for the reception of the sedative solution.

$$\text{℞}\quad \left.\begin{array}{l}\text{Tr. iodi etherialis, acidi carbolici c. p.}\\ \text{Ol. amgydal. amaris}\end{array}\right\}\ \bar{\text{a}}\bar{\text{a}}\ldots\ldots\ 4.0 \text{ gms.}$$

Spts. chloroformi q. s. ....ad 32.0 "

of which 10 to 20 drops are prescribed, and the respirator is worn as often and as long as may be necessary.

Should this and other simple means not prove sufficiently sedative some internal remedy may be ordered. Codeine, grain $\frac{1}{2}$ to 1, or heroin, grain $\frac{1}{12}$ to $\frac{1}{6}$, is better than morphine or crude opium because not so likely to cause unpleasant after-effects.

Night Sweats.—In incipient and early cases this symptom is usually allayed by the open-air and hydropathic treatment recommended. In advanced phthisis, on the contrary, perspiration is often so profuse and exhausting that some additional treatment is required. It is my custom to advise first so simple a measure as the sponging of the surface of the body in a solution of vinegar or dilute acetic acid in water (1 to 6) or of quinine in dilute alcohol (1 drachm to the pint). These failing, picrotoxin is prescribed in the dose of $\frac{1}{80}$ of a grain two hours before the expected sweat. Occasionally this dose is given in the mid-afternoon and a second one in the evening.

If the symptom is still refractory camphoric acid is ordered in 15-grain doses t. i. d. after meals, in conjunction with capsicum to aid the stomach in its digestion of the acid. This remedy is often highly effectual but is apt to occasion gastric distress. I very rarely prescribe atropine because of its retarding action on digestive and other secretions, but it is often very efficient in its control or diminution of the perspiration.

The dose of the atropine may be .0005 ($\frac{1}{130}$ grain) to .001 ($\frac{1}{65}$ grain) at bedtime. Agaricine .005 to .01 ($\frac{1}{12}$ to $\frac{1}{6}$ grain) is also recommended, but has not been successful in my hands. Dusting powders of talcum containing salicylic acid, or tannoform (Fraenkel) or alum are also recommended. But whatever agents are employed their action is likely to be short-lived and hence must be assisted by care on the part of the attendant that the bed covering be not too heavy and the atmosphere of the room not too warm.

Fever.—It is in the stage of mixed infection when softening and excavation are proceeding apace that this symptom may call for special therapy. In a considerable number of cases it will be noticed that after several days of absolute rest in the cool open air the temperature range shows a decided tendency to reduction. If such be the case it is well to await the further effect of this simple plan of management. If, on the contrary, this symptom prove refractory or diminish but slightly then it is well to try the effect of sponging, the same as in any other form of fever.

Antipyretic remedies of the coal-tar series are undoubtedly efficient in the reduction of pyrexia but in the tuberculous are apt to prove depressing and to intensify the sweating. Only the least dangerous are at all permissible in this disease and even these in small doses. I think I have never used them in consumption. Dettweiler told me he was in the habit of prescribing phenacetin grains 1½ shortly before mealtime to patients whose temperature went above 101.5° F. because of his belief in the inhibitive action of high body-temperatures on the secretion of free hydrochloric acid. His experience had shown that if the fever could be thus lowered the patient was able to take and digest food. The painting of the skin of the chest with 7–10 drops of guaiacol will sometimes bring down the temperature—and sponging may be resorted to the same as in any other febrile affection. The fever of mixed infection may be controlled by an antistreptococcic serum.

Difficulty of expectoration is occasionally complained of by tuberculous patients particularly in the stage of excavation. Under such circumstances it is well to attempt to facilitate it by simple means but not by ordinary expectorant remedies. *No therapy can be worse than the prescribing of cough syrups and other expectorants.* A hot draught on waking, as hot milk containing half an ounce of rum, will sometimes aid in the raising of sputum. Carbonate of ammonia or an alkaline water is also useful. A Preissnitz bandage wrung out in tepid water and placed about the chest at night will often facilitate expectoration in a most gratifying manner. I think the inhalation of the drops recommended above on a respirator will also loosen expectoration. The inhalation of steam or the use of a nebulizer after the plan advised in the treatment of acute or chronic bronchitis is far preferable to internal expectorant drugs.

Chest pains, especially such as result from dry pleurisies, call for relief in some instances. Opiates should not be prescribed but anodyne liniments the ice-bag and strapping may be tried and often prove all that is necessary. Sinapisms and hot poultices may be applied if thought likely to answer better than the other applications.

Digestive disorders sometimes require special medication. If the tongue is foul and the patient has been consuming considerable quantities of nourishment, calomel in divided doses ($\frac{1}{10}$ grain) hourly for a number of hours is likely to do great good. It should always be remembered, however, that tuberculous patients are apt to respond easily to laxative drugs and are often much weakened by strong catharsis. Simple digestants, as extract of pancreas, dilute hydrochloric acid or an alkali in common with a
extract of nux vomica or powdered rhubarb may correct the di
after a few days. If not, then it is well to revise and simplify th
or even to restrict the invalid to milk and an alkaline water fo
four to forty-eight hours. Let it be borne in mind always that
medicine the better for tuberculous patients in whatever stage is a

Constipation is a very common symptom in incipient and e culosis, whereas in more advanced cases there may be an opposite condition. If the bowels are habitually confined and do not become regular by the use of fruits and coarse vegetables, whole wheat bread, etc., then it is well to order a laxative, as cascara. In all instances, however, it is better to try first the effect of olive oil in half-ounce doses at bedtime, the oil being suspended between two layers of orange juice, or a little fig and senna paste, etc. Massage of the colon may overcome the constipation in some instances.

Diarrhœa is a symptom which, in advanced cases or in an earlier period of tuberculosis when obstinate, always excites apprehension lest it betoken intestinal lesions and lead to serious weakness. It is to be treated as in any other disease, by astringents, antiseptics, etc., and diet. I once knew a patient wonderfully relieved of an acute intestinal fermentation with diarrhœa by the exhibition of a 3-grain pill of salol every hour. A physician, himself the victim of pulmonary tuberculosis, once told me that enteric pills of creosote had proved more efficacious in his case than any other remedy he could find. Salophen, tanalbine, subgallate of bismuth tannogen, tannate of quinine may all be tried separately or in combination in various ways.

Hæmoptysis.—This symptom which in some cases is so formidable as to prove a grave complication has been considered in a separate chapter. To this the reader is referred for its treatment.

Complications must be met and treated as they arise. In particular tuberculosis of the larynx must receive especial attention. Most general practitioners are not skilled in its treatment and when such is the case they should not hesitate to call on a laryngologist to afford the sufferer what relief he can. Pleurisy with effusion and empyema will receive special consideration in other chapters and to these the reader is referred.

Drug Therapy.—This part of the subject of phthisiotherapy cannot be passed by in silence because it forms the sole means of management in the hands of a very large percentage of the medical profession. It will be considered not for the purpose of its indorsement but to condemn it in the most emphatic terms possible. Early in my professional career I in common with many others knew no better, but having gained a modicum of wisdom from experience and observation I now desire to make what amends I may by urging my fellow practitioners who still adhere to old-fashioned and pernicious methods to discard drugs, for the hygienic and dietetic management herein advocated.

The stomach is the consumptive's citadel! Destroy his digestion and he is a lost man! What therefore can be more pernicious than the administration of cough syrups, tonics, compounds of creosote and peptonoids or guaiacol and hypophosphites, etc., with which the market abounds! They are all prescribed in the hope of improving the appetite and thus increasing nutrition, but instead of accomplishing the end desired, in the bulk of cases they destroy what little appetite was left, and derange digestion. Drug therapy is the last resort of the ignorant and the doom of the consumptive!

The Use of Creosote and Guaiacol or their Derivatives.—It seems to me the history of the use of these remedies forms one of the sad chapters in medical literature. Creosote seems to have been introduced to the notice of the profession by a monograph published by Stoehr, of Germany, in 1848, but it did not gain wide popularity until the indorsement of Bouchard and Gimbert, of France, in 1877, and the enthusiastic claims of Sommerbrodt in 1887. The last mentioned believed that if taken in as large amounts as the digestive tract could be made to tolerate, creosote became an actual germicide in the blood. It was in this belief also that Gimbert had previously advocated its administration hypodermically in a sterilized olive-oil emulsion.

It is now claimed for creosote and guaiacol that their beneficial action is local, serving to improve appetite and digestion and to disinfect the intestinal tract. They are known also to exert a mild expectorant effect and perhaps by reason of their partial elimination through the lungs to exert a mild antibacillary action.

It is mainly for the sake of favorably influencing appetite and digestion that they in their crude form and their numerous offspring Duotal, Benzosal, Geosot, Kreosotal, Tunosal, Phosot, Tuphsot, Phosphotal, Eosot, Sirolin, Thiocol and perhaps more, are so widely advertised by disinterested (?) pharmaceutical chemists. So extensive is their administration under the dictation of the commercial houses handling them, that one can scarcely go along the streets without having his nostrils assailed by the penetrating almost nauseating odor which marks the consumptive leaning on this broken reed.

Very exceptionally a tuberculous individual is found who can tolerate pure creosote in enormous doses, and truth and candor compel me to say that such a person may receive benefit therefrom. In the overwhelming majority of persons, on the contrary, the habitual ingestion of even carbonate of creosote or carbonate of guaiacol, both of which are supposed to be unirritating to the stomach, produces unpleasant eructations, gastric uneasiness and, in larger doses, even vomiting and diarrhœa. Instead of improving the appetite and digestion these various drugs exert a contrary effect.

There are innumerable circulated reports which attest the salutary effect of these agents on consumptives and it is these reports which capture both doctor and patient. But where are the thousands of cases of persons who have tried in vain hope to take these creosote preparations and been forced to desist? They remain silent, yet it is these cases of failure that condemn creosote, and not the smaller number of successful cases that should recommend it. In comparison with the hygienic and dietetic plan o
agement now everywhere achieving such hopeful and brilliant res
use of creosote and guaiacol and their derivatives deserves to sin
oblivion.

The Use of Cod-liver Oil.—This is another relic of a bygone tim
faith was pinned to the use of internal remedies. Its employment w
on the need of the consumptive for fat in an easily digested form

the fact that it was believed to contain free iodine which with other ingredients made it the ideal remedy for this wasting disease.

The class of cases for which this oil is suitable are those in the chronic stage without fever and digestive weakness. It should not be prescribed hit or miss, as it were, but with a clear understanding of its advantages and contraindications. It is not well borne when there is much pyrexia or in hot weather. It is very apt to occasion unpleasant eructations and to interfere seriously with appetite and digestion.

As the odor of the oil is a serious objection to its use by sensitive patients, only a purified pale preparation should be ordered since the dark-colored oils are no more efficient and contain ingredients very likely to disturb the stomach. Cod-liver oil may be suspended between two layers of cold coffee or wine or in an emulsion, when it is far more apt to agree. The initial dose should be small (one to two teaspoonfuls) and increased as a tolerance is established. After meals either directly or about an hour is the best time for its administration, though occasionally a person can take it just before retiring for the night to better advantage.

Cod-liver oil should never be persevered with if it deranges digestion since it will do more harm than good and its place as a fat can be supplied to advantage by cream, butter and possibly other forms of fat.

Hypophosphites.—Another group of drugs on which much reliance is placed by many is a syrup of the hypophosphites of lime, soda and potash, particularly the first. Its use was advocated on the assumption of its being a powerful factor in calcification of tubercles and thus in the arrest of this pulmonary disease. In some cases of the chronic variety in which a tonic seems indicated a hypophosphite preparation may be of service if prescribed for a short time but it cannot replace food and fresh air, even in these cases. The syrup, because containing sugar, disagrees with some persons and if the relish for food lessens during its administration the medicine should be at once discontinued.

Malt extracts are still another therapeutic weapon which some practitioners wield in their vain onslaughts on this disease and which are recommended because of their nutrient value. They are usually found in the form of emulsions together with hypophosphites or oil or both. All these remedial agents may prove beneficial in certain cases, namely, chronic ones characterized by emaciation and weakness rather than by septic symptoms, but they are doubtful aids in the combat against tuberculosis and are better left alone if they in any wise interfere with the patient's ability to consume large amounts of food.

Other tonics as arsenic, iron, strychnine and the like may be prescribed in chronic cases displaying much anæmia, but are distinctly inferior to life in the open air, sunshine, exercise and food in the building up of the blood and tissues of the body. *Strychnine* in particular has been advocated in as heavy daily dosage as the patient can be made to tolerate, on the assumption that a vice of the nervous system is at the bottom of pulmonary tuberculosis. This agent, as well as other bitter tonics, may stimulate the individual for a time and arouse a flagging appetite, b [redacted] ikely to produce just the opposite effect of what is desired. *In fact, the routine and persistent administration of this or any other drug is what is especially deprecated, not their occasional, intelligent use.*

Climatotherapy.—The discussion of phthisiotherapy would be far from complete were the consideration of a change of climate neglected. Of late, under the influence of the sanatorium idea, there has been growing up in certain quarters an inclination to scout the benefit or desirability of a change of climate in the treatment of pulmonary tuberculosis. It is argued, in effect, that since it is life in the open air, together with other hygienic and dietetic accessories, which accomplishes a cure either absolute or relative there is nothing to be gained in one climate over another.

While agreeing fully with the premises of the proposition I cannot coincide with the conclusion. As regards most health resorts I believe their chief recommendation lies in the opportunity they afford for life in dry, pure air and sunshine at an agreeable temperature, free from the sudden and extreme changes that complicate the problem at home. While dryness of the air is not indispensable to the recovery from this disease, as witness the results obtained in Germany, New England and Great Britain, still it is an important factor in rendering these sensitive patients tolerant of both heat and cold, and exerts a beneficial influence upon the catarrhal condition accompanying the tuberculous process. Dust is a necessary accompaniment of lack of moisture and is a decided drawback to some resorts. The same thing can be said of the winds which often whirl clouds of dust along with them. For these and other reasons I do not regard great dryness of the air as an unqualified recommendation.

Purity of the atmosphere is assuredly a great desideratum and furnishes a very strong argument in favor of some resorts as compared with the home climate, and yet it must be acknowledged that the air in a sparsely settled locality is not so contaminated with germs, dust or smoke as to render it injurious for the tuberculous. It is the abundance of *sunshine* and the great preponderance of *cloudless days* that make most of the health resorts so desirable a place of residence for consumptives. Dryness and sunshine go together very largely, and hence the former with the consequent dust and wind sweeping over the open stretches of country is a necessary accompaniment of sunshine and freedom from rain and snow. The effect of this combination of dryness and sunshine is to make the climate exhilarating and agreeable to most tuberculous individuals, notwithstanding the irritation of the mucous membranes of nose and throat sometimes found very disagreeable.

Freedom from sudden changes of weather is a special recommendation of certain climates since the invalid can reckon with certainty on pleasant weather from the beginning of a day to its close and often from one end or one month's end to another. Consequently, in such an equable the tuberculous invalid runs comparatively little danger of the cong and catarrhs so dreaded in the open-air management of these patients changeable, often cloudy and damp climate at home.

Accordingly any climate which possesses the qualities above stated especial facilities for the treatment of pulmonary invalids. There other phase of the question still to be considered, namely, altitude. Does a mountain climate offer special advantages to the tuberculous? It is my opinion that it does! For a detailed discussion of the special features and influences of such a climate I must refer the reader to such works on Climatology as that by S. E. Solly, of Colorado Springs, whose experience and knowledge entitle his views to marked consideration. It is sufficient here to direct attention to the stimulating effect of mountain climates on the blood-making organs, to their influence in causing expansion of the lungs and to their tonic effect in general, as well as to their possession of all the qualities of dryness, purity, sunshine and, for the most part also, their equability as compared with seashore or lowland climates in the temperate zone.

Certain other things are absolutely indispensable besides the qualities of the climate, however, and these are *good accommodations and the attendance of skilled phthisiotherapists.* Without these necessities the home climate with all its drawbacks of weather is preferable. Nothing is a greater mistake in most instances than for the tuberculous invalid, particularly if he has passed the first stage, to go for a change of climate to some so-called health resort where he cannot obtain good nutritious food and where he knocks about without the guidance and oversight of a physician expert in the management of such patients.

My position with reference to a change of climate is the following: (1) A person with incipient or early tuberculosis of the lungs, particularly if limited in extent, can ordinarily be treated successfully at home by a physician who understands the subject. (2) If his circumstances permit he would better be sent to a closed sanatorium managed on the most approved principles. For certain individuals the restraint and discipline of such an institution are indispensable to a cure. (3) If he will not or cannot go to a sanatorium then send him, if he have the finances, to some health resort, by preference to a mountain resort, but make it a *sine qua non* that he place himself, when there, under the care of a phthisiotherapist of recognized skill, experience and integrity. (4) Give over the conduct of the case to the expert to whom the patient is referred. (5) If the case be one with pronounced constitutional disturbance and local changes indicative of acute or rapidly progressing tuberculosis it should be kept at home or in the vicinity of the home under the management of the family doctor, or of another physician if the former does not understand the hygienic and dietetic treatment of the disease. (6) The transfer to another climate at a later period must be determined by the condition when that time arrives. (7) If the case be one of chronic pulmonary tuberculosis without severe symptoms it would better be sent into another climate, but under the conditions believed essential in an incipient case. (8) If the finances do not admit of the consumptive's procuring suitable food and accommodations or availing himself of the care of an expert physician, then he would better remain at home. (9) If the case is chronic but rapidly progressing it is not well, ordinarily, to send it away, since the chances are that the patient would not recover and would be injured by the journey.

Choice of Climate.—The question that next arises, after it has been decided to send a patient from home, concerns the choice of a resort. As a general proposition the selection of a climate is of less moment than is the likelihood in any given place of good quarters and skilled medical attendance. Assuming that these are satisfactory the individual condition must be considered. A person with incipient disease still closed, that is, not discharging bacilli in the sputum, and in fairly good physical state may go to almost any resort where he can be outdoors day and night. I should prefer a climate that is not too hot and damp nor subject to atmospheric changes, believing that the colder the climate which the person can bear and yet carry out the hygienic plan of treatment, the more likely his recovery and the more permanent his cure. A subsequent return to his native clime will be less likely to prove harmful. For this class of patients I prefer Colorado or New Mexico or the resorts in the valley of the Engadine. They may get on well, however, in California and those elevated parts of Arizona, Mexico and southwestern Texas where conditions of living already designated can be secured. Many of them do well also in parts of North Carolina, South Carolina, Georgia and even Florida.

Persons in the first stage or in that phase of the disease, when the first is passing into softening, yet without marked symptoms of infection, may be sent to almost any of the climates just mentioned, but if the process is quite active I think it better to keep them at home until it has become less acute. They may seek higher elevations at a later period when their process has become partly arrested. Another contraindication to high altitude is found in extensive pleuritic adhesions or in such an extent of infiltration as to cause considerable decrease of vital capacity and dyspnœa of effort.

Chronic consumptives may be governed in a selection of a climate by some other considerations than merely climatological ones. If they contemplate permanent residence they should, *ceteris paribus*, choose a resort where they will be likely to find congenial society and an environment calculated to further contentment of mind as well as physical well-being. A high altitude is not suitable, however, if vital capacity is greatly reduced or if *hæmoptysis* has recurred. For further elucidation of this subject and discussion of seaside resorts the reader is referred to works dealing with Climatology.

The *combination of closed sanatoria and a salubrious climate* is one that appeals to the practitioner in an unfavorable locality with especial force. Fortunately this combination is rapidly being formed and is to be found in Denver, Colorado Springs and elsewhere. In the selection of such a sanatorium, however, one should be cognizant of and satisfied with its conditions, the same as in the selection of a resort, or open sanatorium as it is called. The name of sanatorium is not always synonymous with excellence regarded from the viewpoint of modern phthisiotherapy.

Treatment by Means of Tuberculin.—Many of us can rec enthusiasm which greeted Koch's tuberculin as a possible means of tuberculosis. We can also recollect the many reports of injury that from its injudicious administration. In consequence of these adverse the profession in general came to look with disfavor upon this the agent and the use of Koch's tuberculin became practically restricted to the diagnosis of obscure cases. There has always remained a limited number of clinicians, however, who have employed the remedy in certain appropriate cases. Of late the interest in this subject has been revived and so many men of trustworthy judgment have reported favorably upon its proper use that this chapter would not be complete without a discussion of this means of therapy.

Action of Tuberculin.—This substance which is a glycerine extract of pure cultures of the tubercle bacillus excites an inflammatory reaction about tuberculous foci when injected beneath the skin of animals. This reaction is evinced by fever, *malaise*, nausea and even vomiting, while locally changes in the lungs are produced which manifest themselves by râles. *If the dose is too powerful or if regardless of the reaction* the subcutaneous injections are continued, there is created a necrosis of the tuberculous areas with formation of cavities and at the periphery a further increase of the infiltration. These effects have been demonstrated both clinically and at the necropsy.

If the tuberculin is administered in *sufficiently attenuated doses and increased cautiously* so as to avoid febrile reaction, the local inflammatory changes may, it is asserted, lead to formation of fibrous tissue which shuts off the tuberculous focus.

Some individuals are found to react to smaller doses than do others, but in all cases there at length comes a time when, if the injections have been increased slowly enough, they will no longer occasion reaction. This is taken to indicate the acme of beneficial effect and the treatment is stopped. A still further proof of the curative action is found in diminution of sputum, with disappearance of bacilli from the expectoration, and in gain of weight.

Still another index of the beneficial influence of tuberculin injections is found in the effect upon the opsonic power of the blood. By this term is meant that property in virtue of which the phagocytes possess the power of destroying and incorporating micro-organisms. This is, of course, a part of the machinery of defense against infection. In healthy individuals the opsonic power of the blood is uniform, whereas in those who are tuberculous this power fluctuates from day to day (Urwick) and is always low. If now tuberculin injections produce a beneficial effect it is found that the opsonic power of the blood is increased. Wright has found, however, that if tuberculin is to do good it must be injected when the opsonic power of the individual is on the increase and not on the decrease. Otherwise the effect of tuberculin is harmful.

Since the action of tuberculin is limited to the tuberculous process the remedy is considered unsuited to cases of mixed infection or at all events is more efficacious in early cases. Some therapists employ the agent in the second stage, but only when fever is absent. In this stage, moreover, the best results are said to be obtained when there is a tendency to fibrosis.

Methods of Administration.—Experience has demonstrated conclusively that, if harm is not to result, tuberculin must be given in more minute doses than were originally recommended by Koch. Accordingly, such experienced clinicians as Goetsch, Romisch, Moeller, Löwenstein, Rappoport, Petruschky and others begin with exceedingly attenuated doses. Koch's old tuberculin is diluted with an 0.5-per-cent solution of pure carbolic acid and of this so small an amount is injected as in all probability will not occasion reaction in even highly susceptible persons.

Goetsch's method is to commence with injections of 0.00001 gm. of old tuberculin and to increase with such caution as precludes the possibility of the slightest reaction until 1.0 ccm. has been reached. The period of treatment lasts, therefore, from 132 to 791 days according to the susceptibility of the patient. If so small an amount as 0.00001 gm. is not borne without reaction, a preparatory course of treatment is instituted with Koch's TR which, beginning with 0.001 mg., is gradually increased until 0.1 mg. of tuberculin R. has been attained. Thereafter it is generally found that the ordinary initial dose of old tuberculin can be endured.

Goetsch reported that with TR alone he had never been able to secure a disappearance of bacilli from the sputum although he had gone to as high as 20 mg. By the combined treatment as just described he had succeeded, however, in the removal of the germ from the expectoration. He treated only afebrile cases which showed no extensive excavation and therefore would be likely to manifest good results if they were to be restricted to the hygienic plan of management alone.

Roemisch's method is similar and with it he has reported good results in persons who manifested severe tuberculosis. He begins with TR but with an initial dose of $\frac{1}{500}$ mg. of the TR and increases until 0.1 mg. is reached. He then begins the use of old tuberculin in minute doses and increases up to 1.0 which amount is repeated several times.

In the light of his results Roemisch recommends this plan of treatment for such advanced cases as have not been benefited by other approved modes of management, provided the patients have no fever, are not very nervous and do not show signs of extensive destruction of lung tissue, and provided the tuberculous infiltration is not very widespread. In this latter event a possible reaction to tuberculin might produce serious consequences. Those patients are especially likely to be benefited who show pulmonary changes of a fibrous nature.

Moeller and Kayserling lay stress on the circumstance that the prospect of improvement is not to be measured by the pulmonary condition alone but by the state of the general health, those patients being the most likely to receive benefit who present favorable local and constitutional conditions.

Petruschky's method is said to consist in three or four repetition
tuberculin treatment after intervals of several months. The initi
tion is $\frac{1}{10}$ of a milligramme or even less and this amount is rapidly i
until the individual manifests slight reaction. From this time on,
crease is gradual until no further reaction can be occasioned. An
of months is now allowed to elapse, after which the course of inj
resumed in the same manner as at first. Thus three or four
treatment are given

Petruschky and Weicker claim that if tuberculin is administered in this manner it establishes a cure and this is shown by the failure of the individual to react to a diagnostic injection of tuberculin ten to twelve months subsequently. They recommend this plan of treatment in combination with the sanatorium management and especially for persons who are not able to submit to an institutional management for an indefinite length of time.

Wright's method is based on the state of the opsonic power of the blood in each case and includes the use of new tuberculin. Before giving an injection the opsonic power is tested and if this is found on the upward curve an amount equal to $\frac{1}{1000}$ to $\frac{1}{600}$ of a milligramme of powdered bacilli is administered. To obtain the opsonic index in a given case a standardized culture of tubercle bacilli is submitted to the action of the individual's blood, and the number of germs that can be taken up by the leucocytes is noted. If the opsonic index is found to be falling the tuberculin is not administered. The results claimed for this mode of treatment are highly satisfactory.

Treatment by Means of Anti-tubercle Serum.—This furnishes another example of hope raised only to be dashed to the ground. During the last decade of the preceding century numerous sera of this sort were exploited but at the present time only two present any claims to attention.

Maragliano's Serum.—This is obtainable in sealed tubes containing 1 c.c., 5 c.c. and 10 c.c. respectively and is administered by subcutaneous injection, as follows: 1 c.c. every other day for 10 days; 5 c.c. every other day for the next 10 days; 10 c.c. every other day for 20 days more, and if no beneficial results are then perceived the remedy is discontinued. The results claimed are improvement of digestion, anæmia, perspirations and fever, increase in weight and improvement in the local pulmonary signs. Disastrous effects are not observed. This serum is suitable for chronic cases showing but very slight pyrexia, or none at all.

Marmorek's Serum.—This does not appear to have been much employed in this country, but finds enthusiastic supporters among those who have given it considerable trial abroad. It is claimed to possess antitoxic properties and Richer, of Montreal, believes he has seen it bring incipient cases to an arrest. The serum may be injected beneath the skin in amounts varying from 3 to 10 c.c. and at longer or shorter intervals over a period of weeks or months, according to the effects. The serum is said to reduce febrile temperature and to cause a clearing up of the lungs. It is indicated in cases of unmixed tuberculosis, especially.

Additional Non-specific Means of Treatment. — *Antistreptococcic serum* is an agent to which physicians have turned occasionally in the hope of combating the septic phenomena marking the secondary or mixed infection. There have been reports of an encouraging nature and further trials of this agent are warranted with a view to proving its value. Nevertheless, in cases showing high fever and profuse perspiration with copious purulent sputa some one of the many sera of this nature may be injected with a view to ameliorating the general symptoms. Administered with caution such a serum may not do harm, and may furnish another weapon in the hands of the phthisiotherapist.

Inhalations of various sorts have been and still are extensively employed in the treatment of this widespread disease. They consist of sprays of many kinds and among these of substances that exert a liquefying effect on the secretions or are supposed to act antiseptically on the various organisms concerned in the causation of consumption.

It is needless to say that whatever be their nature or however powerful their antibacillary action in the laboratory they do not and cannot destroy the bacilli in the lungs. They may be a justifiable adjunct to the hygienic and dietetic treatment herein advocated, but when inhalations are made a pretext for enticing tuberculous patients to one's office they are a delusion and a snare and as such merit condemnation.

The Production of Artificial Immunity.—By immunity against a disease is meant the resistance to that particular infection possessed by the tissues. Accordingly, we distinguish three kinds of immunity: namely (1) natural, or that residing in the organism of the animal by virtue of some peculiar property of its tissues, (2) acquired, or that protection afforded by previous attacks and seen in variola, (3) artificial, or that produced by the introduction into the system of some agent capable of bringing about a resistance which did not previously exist. An example of artificial immunity is seen in the vaccination against small-pox.

It was early recognized that certain animals as the horse, ass, goat, dog, possess a relative degree of immunity against the action of the tubercle bacillus. They are not absolutely proof against this form of infection and perhaps no animals, not even the cold-blooded, are, but it requires either more virulent bacilli or larger numbers of the bacilli to infect some animals than others, e. g., guinea-pigs, or human beings.

It was noted, moreover, that some persons appeared to resist the action of the tubercle bacillus to a greater extent than others and that in nearly all individuals the characters of the tuberculous lesions evinced an attempt at resistance, shown in fibrosis and calcification and by the discovery of healed lesions in a large percentage of all bodies coming to autopsy.

Such observations and facts soon led bacteriologists to entertain the hope that some means might be devised of creating in human beings an immunity either absolute or relative against this widespread scourge, tuberculosis. Now there are two methods of establishing protection which are known as the toxic and the bacteriolytic. The former is represented by diphtheria antitoxin; and as respects tuberculosis is exemplified by the various anti-tubercle sera and by the tuberculin treatment. It has been shown that although the system may be assisted in its struggle against this disease by the use of tuberculin or of a serum as claimed by Maragliano and Marmoreck it cannot be actually protected by these substances against infection. cordingly, some other method of producing immunity is necessary and i tigators turned to experiments looking to a bacteriolytic or isopathic i nity as von Behring terms it. In other words, if the inoculation living bacilli is to prove protective to the animal experimented upo germs introduced must be of diminished virulence so that an inflamm reaction is set up which results in the victory of the invaded tissues o invading bacilli as shown by the arrest and healing of the process first produced by the micro-organisms.

It is not necessary for the purposes of this work to enter into an account of the many attempts that have been made at the establishment of immunity of this kind; it will suffice to record the fact of failure until within very recent years. Among the indefatigable workers in this line may be mentioned Falk, Daremberg, Martin, Grancher, Courmont, Dor, Richer, Hericourt, Koch, McFadyean and Behring in Europe, and Trudeau, Pearson, Gilleland, and De Schweinitz in the United States. Of the last mentioned it is Trudeau and his able corps of assistants at Saranac who deserve especial mention. It is to contributions by these men and to personal communications from E. L. Baldwin that I am largely indebted for facts embodied in this short account.

The plan of work quite generally adopted is the inoculation of animals with attenuated cultures of living bacilli of human origin and then, after a month or so, the introduction into these same animals of a small amount of virulent cultures of the tubercle bacillus. For details as to technic, dosage, etc., the reader is referred to the original papers of the workers named. In 1894 De Schweinitz announced the production of protective immunity in guinea pigs, a cow and a calf. In 1902 von Behring published an exhaustive account of his success in immunizing calves by means of attenuated cultures of human bacilli and in the same year L. Pearson and G. H. Gilleland reported their success in protecting cows by inoculation with living cultures of bacilli obtained from human sputum. It will be noted that in these instances the animals experimented upon were very susceptible to this particular infection but were large and did not present some of the difficulties and sources of error found in experimentation of guinea pigs and rabbits which are the animals almost exclusively used at Saranac. If, therefore the results obtained by Trudeau and his coworkers are not so decided they are nevertheless characterized by such conscientiousness and such a scientific spirit that the conclusions to be drawn from them are none the less valuable.

Before summarizing their results it will be well, however, to turn for a moment to some work being done along a somewhat different line. Trudeau and nearly all the workers in Europe and in this country have carried on their investigations with attenuated human bacilli or germs of avian origin, that is, with bacilli obtained from warm-blooded animals. On the Continent of Europe experiments have been carried out with germs obtained from cold-blooded animals or that had been passed through cold-blooded animals. The results as briefly stated by Baldwin are as follows:

Terre failed to produce any marked protection in a guinea pig by means of the fish bacillus cultivated from a carp. Moeller obtained a high degree of immunity in guinea pigs and rabbits against the human bacillus by means of the slowworm bacillus cultivated from such a worm which he had inoculated with sputum. This culture grows only at a room temperature (22° to 25° C.) to which fact Moeller attributes its lack of virulence. It resembles the fish bacillus of Batallion, Dubard, Moeller and Terre.

Such was Moeller's faith in the nonvirulence of these bacilli that he inoculated himself with these bacilli subcutaneously and intravenously, and only a transient disturbance followed. Three weeks later a small intravenous inoculation with human bacilli had no apparent ill effect, except that in the next two months he lost fifteen pounds in weight which, however, were soon regained.

Dieudonné passed mammalian bacilli through frogs and then, after frequent growth in cultures at 25° to 30° C. which remained the maximum temperature, he experimented with guinea pigs immunized with these cultures and afterwards inoculated with virulent mammalian bacilli. The result was variable. Some pigs suffered only slight infection, others died at the same time as the controls or a little later. Friedmann experimented extensively with cultures from spontaneous lung tuberculosis in a turtle which was probably of mammalian origin. They resembled mammalian bacilli in all respects save in virulence, producing at the worst only localized nodules which healed. Various animals, guinea pigs, rabbits, cattle, goats, swine, etc., were claimed to be completely protected by previous intravenous inoculation of turtle bacillus cultures. The serum of cattle and swine thus immunized was also claimed to be partially protective to guinea pigs.

Libbertz and Ruppel who witnessed and controlled some of Friedmann's experiments refuted most of his claims, but admitted a retarding influence on the disease in guinea pigs by means of the turtle bacillus inoculations.

Returning now to the work done at Saranac it may be stated that E. L. Baldwin has received cultures of Moeller's fish and slowworm bacilli and that these have been experimented with in Trudeau's laboratory. These were reported by Trudeau at the Washington meeting of the National Association for the Study and Prevention of Tuberculosis in May, 1905, and were subsequently published in the Medical News for September of that same year. They were a part of a series of experiments carried out to determine the immunizing value of old cultures of human bacilli, and hence an excellent opportunity was afforded of comparison between these and those obtained from Moeller and with which he claimed such brilliant results.

That the reader may obtain a clear understanding of the results it has been decided to quote at length from Trudeau's address mentioned above.

"I determined to make an experiment that would be likely to shed light, (1) on the degree of protection afforded by cultures of warm-blooded origin but attenuated by prolonged growth, as compared with that conferred by bacilli derived from cold-blooded creatures, and which apparently die at once when introduced into warm-blooded animals. (2) To determine whether the degree of attenuation obtained by prolonged growth bears any relation to the degree of protection afforded; that is, whether a culture of hum
grown for over twenty years on artificial media, which produces now
no appreciable local lesion and never tends to generalize itself, will
well as one also of human origin which has been cultivated for over
years, and which still produces in all the guinea pigs slightly enlar
nal nodes near the site of inoculation, and occasionally even—t
about one in ten animals—brings about a little caseation in suc

with a tendency to become generalized to the extent that bacilli in a few instances reach as far as the spleen. The former culture is designated K 1, and is one of the original cultures which Dr. Koch separated in his first work on tuberculosis. The second is my old R 1 culture, which I obtained from the human subject, and has grown, principally on glycerine peptone bouillon, about fifteen years.

"On February 27, 1905, 48 guinea pigs of the same size were separated into four groups, and each group received respectively, in the left groin, one-half a milligramme of each of the following vigorously growing agar cultures: Lot I, human R 1 culture; Lot II, human K 1 culture; Lot III, cold-blooded (frog), and Lot IV, cold-blooded (slowworm) culture. A month later one of each group was killed. Slowworm and frog guinea pigs showed absolutely no lesion. K 1 animals show enlarged nodes, no caseation or tubercle bacilli. R 1, one or two enlarged nodes, slight caseation and a few tubercle bacilli. No other lesions anywhere. The animals were all tested with tuberculin, and only the Lot I, human culture R 1 pigs, gave any reaction.

"March 27th, thirty-three days after vaccination, all the vaccinated animals, together with twelve controls, were injected subcutaneously in right groin with one-tenth milligramme virulent human tubercle bacilli culture. All the 55 animals were killed at the same time, May 10th, or 44 days after the virulent inoculation, and having been divided in five lots, were laid out side by side for comparison. The animals of each lot were quite uniform in the character and extent of their lesions.

"*Lot I, Vaccinated R 1 Human,* show enlarged and, occasionally, cheesy nodes, slightly enlarged spleen, abundant perirenal fat. No other macroscopical lesions are visible, no tubercles or caseation.

"*Lot II, Vaccinated with K 1 Human,* show caseous nodes and much larger spleen (over twice as large as Lot I); absence of perirenal fat marked in nearly half the animals, but no caseous tubercles, though a few gray tubercles could be seen in liver and lung in about one-third of the animals.

"*Lots III and IV,* vaccinated with cultures from cold-blooded animals (frog and slowworm), as well as the controls, all show to about the same degree the usual picture of well-advanced generalized tuberculosis in the guinea pig. Spleens about three times the size of Lot II, riddled with gray caseating tubercle. Entire absence of perirenal and lumbar fat, cheesy areas in liver, and gray tubercle in the lungs.

"The controls and animals of Lots III and IV were in no way distinguishable, while any one of the animals, Lot I and Lot II, could have been picked out easily if thrown among controls, or victims vaccinated with cultures from cold-blooded animals.

"This experiment seems to offer some interesting evidence. First, there is evidently a relation between the degree of protection and the attenuation of the culture used as a vaccine. The R 1 human, which from its production of local effects, enlargement of neighboring nodes containing bacilli, and slight tendency to generalization (bacilli having been found occasionally in the spleens of animals injected with this attenuated culture), protects better against subsequent virulent inoculation than the K 1 culture, which produced hardly any appreciable and purely localized effect, no bacilli being found to have penetrated even the inguinal glands near the inoculation spot.

"The frog and slowworm bacillus, which causes no local disturbance at all, seemed to have no effect in protecting the vaccinated animals, for the lesions of these exactly resembled those of the controls. The conclusions to be drawn from both of these experiments would therefore seem to be, (1) that dead tubercle bacilli increase, though to a very slight degree, the animal's resistance to subsequent inoculation; (2) that the living attenuated bacillus gives a stronger degree of immunity than the same bacillus killed by heat; (3) that the degree of attenuation of the bacillus used as vaccine bears a distinct relation to the degree of protection it affords in guinea pigs to subsequent inoculation with virulent human cultures. That a culture still capable of producing a very small amount of cell destruction, and of spreading to the neighboring inguinal nodes, gives better protection than one which produces hardly any appreciable and purely localized tissue changes; (4) that cultures derived from cold-blooded animals and which grow only at room temperature, as used above, have brought about no appreciable degree of immunity; (5) the chemical changes produced in killing the bacilli by heat in the first experiment cannot wholly explain the lack of protective power of the vaccination with dead bacilli, for the K 1 human bacilli used in the second experiment, though they had not been killed by heat, failed to give as good protection as the R 1 human culture, which differed from it only in the degree of its virulence.

"The evidence here presented would seem to be in keeping with what we know of the artificial immunizations. Toxin immunity, or immunity brought about with dead germs, is never as strong or as lasting as that produced through the medium of a living virus (passive and active immunity). Furthermore, the degree of the attenuation of the virus greatly influences the degree of immunity obtained."

This subject may not be dismissed without a condensed statement of the report made by Baldwin at the Washington meeting in May, 1905, concerning the agglutinating and opsonic power of the blood of vaccinated animals.

The vaccinated animals (hares and rabbits) and the controls were all killed by bleeding under ether and the serum was tested either on the same day or twenty-four hours later. It was found that a pulverized bacillus emulsion (prepared by diluting one part of 1 in 1,000 Koch emulsion to three volumes with .85 per cent NaCl. solution which made a weak opales-
cence) was precipitated from the third to the thirtieth day afte
inoculation by the serum of the vaccinated rabbits in dilutions o
1 in 10; 1 in 15; 1 in 20; or higher when the serum proved a
the controls whose serum was also tested 9 gave a similar ag
reaction but as 6 of these 9 controls were found to have coccidi
liver it was thought the agglutinating power of the serum in the
to this complication. The remainder of the controls did not

specific reaction to Koch's emulsion and thence the results in the deemed fallacious and were thrown out. The conclusion seemed j therefore, that in vaccinated animals the blood acquires the power c festing a specific reaction against the bacillus if indeed it be not of combating the germ.

Baldwin and his coworkers also studied the power of the ph in taking up both living and dead bacilli according to the method by Wright and Douglas in respect to the serum of tuberculous undergoing successful tuberculin treatment and known as opsonin consists in counting the number of bacilli contained in a number nuclear leucocytes after the method of Leishman in stained specimen method is very difficult in rabbits on account of the small size of th cytes and hence the investigators were compelled to modify it so It was found that the opsonic power of the blood of vaccinated was not increased, and in this respect the serum furnished the opp what was demonstrated with regard to agglutination and sedim Accordingly, Baldwin expresses the opinion that this want of un in results will require further investigation.

On the whole, therefore, it can be said that it is possible to pro tain of the lower animals, e. g., calves absolutely and certain others bits and guinea pigs relatively or partially. Hence it is to be hop the time will yet come when it will be possible to immunize children tuberculosis. But, as remarked by Trudeau in a paper read in 190 the Association of American Physicians, we do not yet know w dangers of immunization are, or how long such artificial immuni last. The inoculation of animals with virulent cultures of tubercl is a very severe test and it is to be hoped that since human bei hardly likely to become infected in so virulent a manner the establ of an artificial immunity might be accomplished. Behring has s that the milk of immunized cows might be made use of for immunizi dren, but if such a trial be ever made years will be required before can be considered conclusive.

The results claimed for the tuberculin and for serum therapy disease also suggest that possibly a complete immunity will have to toxic and bacteriolytic. At all events enough has been accompli demonstrate that in this line of research lies the future solution mighty problem. Scarcely had the foregoing paragraphs been writt von Behring made his startling announcement that he had successf munized calves and believed he could do the same with human bein these pages are going to press he has begun to distribute for expe purposes his immunizing substance under the name of Tulase.

CHAPTER XXXI

FUNGOUS DISEASES OF THE LUNGS

I. ACTINOMYCOSIS

It has long been known that cattle are sometimes affected with a certain disease which, beginning in the tissues of the mouth, is called in English Lumpy Jaw. But that the same disease caused by the ray fungus or actinomyces could attack human beings was first made known in 1845 through a case of vertebral caries and abscess reported by the eminent German surgeon, Langenbeck. This was followed in the course of years by similar reports by Lebert, Bollinger, Ponfick, *et al.* Since 1878 the disease has been extensively studied as it manifests itself in man until by now actinomycosis has come to be recognized as of clinical as well as pathological interest. It may affect the tissues of the mouth and throat but is by no means restricted to these parts, being found in various viscera as the liver, and those of the thorax.

In this work we are concerned with the disease only as it appears in the pleura and lungs. In them it may occur in either of two chief forms, the primary and the secondary. The latter is much the more frequent and yet, as Rütimeyer's researches have shown, about 20 per cent of the cases of pulmonary actinomycosis are primary.

In the human being the affection is relatively rare and yet, now that the attention of clinicians has been directed to the likelihood of its occurrence, cases are every now and then being recognized which, up to a few years ago, would have been overlooked. That the disease is growing in frequency in this country or perhaps is coming to be more frequently recognized is proven by the fact that within the past year three instances of human actinomycosis have been discovered among the patients of the Presbyterian Hospital, Chicago. One of these, to which reference will be made in the following pages, has already been published by Dr. E. Fletcher Ingals. Another is at the present writing under the care of Dr. Frank Billings and will also be briefly described in this chapter. A third case, also in the same hospital, proved to be one of actinomycosis together with malaria of the æstivo-autumnal type, the actinomycosis being an accidental d
Through the kindness of Dr. Elmore E. Butterfield, Instructor in P
at the University of Michigan, I have particulars of a most interest
studied by him at Washington, D. C. For many of the facts cont
this chapter I am indebted to Butterfield's paper describing his case,
as to three elaborate articles that have appeared from the pen of Dr.
Hektoen.

There are many considerations which make it clear that mycotic diseases of the respiratory organs are deserving of more attention than is generally accorded to them. This is particularly true as regards country practitioners, and hence this and the two succeeding diseases are especially recommended to their notice.

Ætiology.—The disease we are now considering is due to the action of the actinomyces or ray fungus. This is an organism which has much in common with the hyphomycetes and with bacteria. It resembles the former in its growth and development and the latter in respect to some of the structural details and also in the mode of action of the pathogenic forms. The group actinomyces is an independent one the same as the schizomycetes and it is highly probable that it contains many genera the same as does bacteriaceæ or coccaceæ.

Like the hyphomycetes typical ray fungi develop from small round spores called conidia into cylindrical threads having a branched or network arrangement. The filaments may be said to have an average thickness of half a micron and do not contain phlorophyll. In their original state these threads are homogeneous and solid like many bacilli and in general they stain in the same way as do bacteria retaining their color with Gram's method. Hyphæ or air threads arise from the surface of the mycelia and by a process of segmentation give rise to the conidia which look like chains of minute round bodies and from these conidia the fungus is propagated.

Actinomycetes grow and develop in such a manner that a mass of radiating filaments is produced which possess genuine branches given off at an acute or right angle. The older the colonies the greater is the number and the complexity of these branching filaments, so that the colony is described as a tangled mass of interfelted threads. The younger colonies generally have a radiating or star-shaped appearance and hence the designation actinomyces. (See Plate VIII.)

Harz and Bollinger in introducing actinomyces accurately described the radiation and branching but laid great stress on the clubbing or bulbous appearance of the filaments. We know now, however, that clubbing is not essential and that it is the radiation and branching which distinguishes this fungus.

It is probable that not all forms of actinomycetes are pathogenic and that such forms as possess this quality are the result of their adaptation to parasitism in nature (Hektoen). Nevertheless, inoculation experiments with typical ray fungi obtained from characteristic granulating lesions appear to prove that many of these organisms are slow to adapt themselves to a new environment. When, however, the organism is pathogenic the changes produced are of a chronic nature and are characterized by the production of granulation tissue with foci of suppuration and by the formation of caseous nodules (pseudotuberculosis).

In this respect and in the fact that some of the pathogenic actinomyces are acid-fast there is a close resemblance to tubercle bacilli. Moreover, a relationship between these two organisms is still further shown by t occasional discovery among tubercle bacilli of forms that are distinctly branching. Hence it has been proposed to change the terminology of Koch's bacillus by giving it a name signifying its fungous nature. Thus Coppen Jones has suggested the term tuberculomyces.

Modes of Infection.—The actinomyces abounds in the air, in water and soil and on the beard or chaff of rye, wheat, barley and oats. They are carried, therefore, in the dust of these grains. These facts explain the preponderance of actinomycotic disease among the herbivora. In the case of cattle it is probable that the fungus is introduced into the tissues of the mouth and throat along with fragments of grain and it is in the period of the second dentition, when the tissues of the jaws are particularly tender and liable to injury that infection takes place. Moreover, the feeding of animals with moldy fodder is especially bad since the fungus grows readily in such a material.

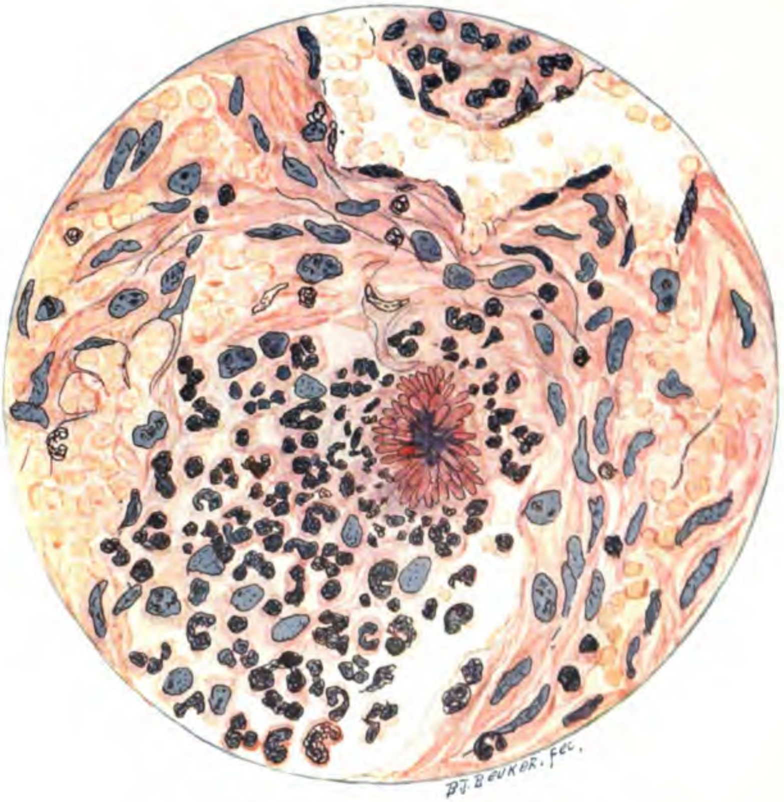
In the case of human beings it is known that infection may occur from contact with the discharges from actinomycotic cattle, but the observations that go to bear this out are not numerous. It is believed, therefore, that the actinomyces gains access to the tissues of the mouth, throat or other parts along with sharp fragments or beard of grain or on pieces of wood that may chance to bear the fungus in their fibers.

In cases of *primary actinomycosis* of the lungs the organism is probably inhaled along with dust or on minute portions of straw or grain. Thus, Balack in the pathological laboratory of Leipzig found a case in which at the apex of the left lung was a small circular cavity of about the size of a bean. Sticking in the fibrous wall of this vomica and projecting thence into its interior was a minute hard object which on microscopic examination was found to consist of a fragment of husk 1 cm. in length and 0.5 mm. in width, and showing on one of its ends a fungus-like body. Lenhartz also mentions the discovery by Ploennigs at Greifswald of a fragment of a head of barley in a bronchus, the lung being surrounded by a dense, fibrous pleura. Such facts emphasize the danger of chewing grains as some persons are in the habit of doing. The relatively frequent occurrence of this disease among millers, farmers, stockmen, etc., is to be explained on the ground of inhalation.

It is believed that the actinomyces finds a suitable culture-medium in carious teeth, since it has been found in a decayed tooth in several instances. I. Israel, who was a pupil of Langenbeck and did much valuable work in this field, published the instance of a coachman in the cavity of whose actinomycotic lung he found a fragment of a decayed tooth. In this case the affection of the lung was primary and the fungus entered by way of the bronchus along with the fragment of the tooth.

In the *secondary* form of this pulmonary disorder the route
may be by the lymphatics or blood-stream. Whereas the latter ro
the occurrence of metastases in the lungs or pleura from the li
where, it is not common. A much more frequent mode of secon
tion is from the tissues of the mouth or throat. It is likely that
the lymphatics the fungus is carried to the tissues of the m

PLATE VIII



Section of Lung Showing Actinomycosis.

In this manner is to be explained the development of prevertebral abscess from which the pleura may become affected.

Morbid Anatomy.—The changes in the lung produced by actinomycosis are essentially chronic and consist of necrosis together with connective-tissue formation. The disease may begin in the bronchi and extend from here outward into the peribronchial tissue, the *broncho-pneumonic* variety, or, commencing in the pleura as a productive or exudative inflammation, it may invade the underlying parenchyma (Fig. 82), the *pleuro-pneumonic* variety. So extensive and complex are the changes, however, that in very chronic cases it is practically impossible to decide to which variety the process belonged originally.

In the *broncho-pneumonic* form the actinomyces causes an initial proliferation of the epithelium of the bronchial mucosa or catarrh which favors its invasion of the bronchial walls and surrounding structures. Necrosis ultimately ensues, and fistulous tracts and small cavities result, which fistulæ run in linear or tortuous directions until the burrowing process may reach all parts and even the parietes.

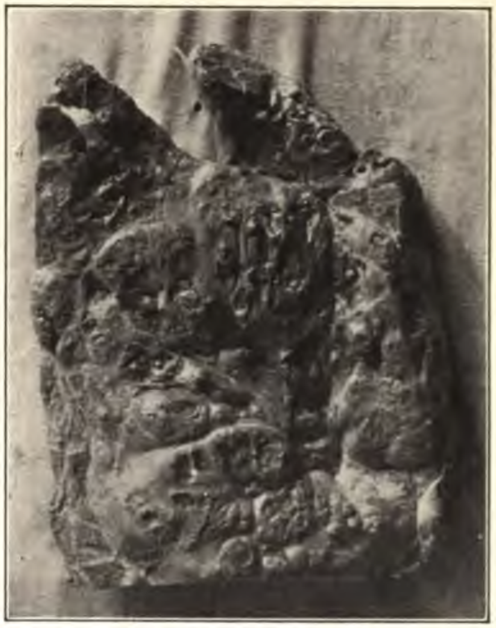


FIG. 82.—Section of bovine lung with actinomycosis.

The lung presents a mottled or marbled appearance, the pneumonic areas being of a reddish or reddish-gray color and the sinuous tracts and cavities yellow or yellowish-gray from the purulent material and fungous granules that fill them. In the course of time the newly formed connective tissue becomes widespread, dense and cicatricial and, undergoing contraction, leads to great shrinkage of the lung, with more or less dilatation of the bronchioles which are also found to contain purulent secretion.

In the *pleuro-pneumonic* variety the inflammatory process starts in the pleura and leads to formation of connective tissue and purulent exudate, so that the pleural cavity is converted into pockets formed by dense adhesions and containing pus of a yellowish appearance. The inflammatory process sooner or later invades the underlying pulmonary tissue causing pneumonia and necrosis in the same manner as in the broncho-pneumonic form. The necrotic process spreads and forms fistulæ and small cavities, fibrous tissue ultimately develops and the picture presented corresponds to that of the other form.

If a cavity be emptied of its more or less yellowish contents its walls are seen to be ragged and soft in the stage of necrosis. The evacuated material is made up of pus cells, masses of fat granules, oil drops, blood cells and actinomycetes, which last are found in the yellowish granules of about the size of a hemp-seed.

The ray fungus attacks all structures with which it comes in contact and hence the necrotic process may extend to the chest-walls forming partially fluctuating or elastic tumors beneath the skin, or it may invade the mediastina, pericardium or heart or, creeping down the mediastinal tissue, may reach the abdominal cavity and any of its contained viscera, especially liver and spleen. It may even reach distant parts of the body.

Although the apex of the lung may be the part first affected as in Balack's case where a small cavity was found at the left apex, still this is not usually the region of predilection. As a general rule the actinomycotic process begins in the middle or basic portion of the lung. It is said, moreover, to affect the left more often than the right lung (Fraenkel).

Butterfield's case is of peculiar interest on several accounts and warrants the quotation verbatim of extracts from the *post-mortem* findings. Its interest lies partly in the fact that the actinomycosis occurred in a pronounced diabetic who died in diabetic coma, and partly, as already stated, in the morphology of the fungus, and last, but not least, in the absence of newly formed connective tissue. It may be added, moreover, that the actinomycetes grew luxuriantly in media containing 2 per cent of glucose, which circumstance, as Butterfield hints, suggests that the glycocæmia favored the growth of the fungus in the lung.

"*Right Lung.*—An area of consolidation about the size of a hen's egg is found in the upper lobe. The pleura over this area is dry and lusterless. On section the consolidated portion is grayish yellow in color, with scattered whitish opaque nodules. There are central disintegration and softening, with the formation of a small cavity of irregular outline and with shaggy walls, which contains a thin, odorless, grayish-yellow, opaque, pus-like material. The remainder of the lung is pale grayish-white, crepitant, with a rather moist cut surface. There is a well-marked carbon deposit."

"*Microscopical Examination of the Pus from the Cavity in the Right Lung.*—Smears were fixed by heat, stained with Ziehl's carbolfuchsin heated to the point at which steam was given off, kept there for five minutes, followed with Gabbet's solution. On examining with $\frac{1}{12}$-inch oil immersion, numerous large masses of tangled bright-red threads are seen on a background consisting of moderately numerous polymorphonuclear leucocytes, large and small mononuclear cells with sky-blue nuclei and colorless or very
light blue protoplasm. The individual threads vary in width fr
three or four times the width of the tubercle bacillus; they
branches which are given off at right or acute angles. No clubs
extremities are observed. While the filaments are usually inter
a colony of indefinite shape and outline, quite frequently a deci
arrangement is demonstrable. The protoplasm of the filaments st
red, but is frequently interrupted by clear spaces, and the free

filaments are almost colorless. On the other hand, a tinge of blue is almost never found in any of the filaments. The colonies are so large that they can be seen easily with a ⅔-inch objective, and the beading of the filaments is so clean cut that it can be seen without any effort through a ⅙-inch lens."

It may be added that "although the interlobular septa and alveolar walls are much thickened, careful staining after the methods of von Giesen and Mallory fails to show an increase in the fibrillar connective tissue." What influence, if any, the diabetes had in preventing the development of connective tissue must be left to conjecture.

Symptoms.—The clinical picture is, in the main, similar to that of pulmonary tuberculosis. There are, however, important differences. Anæmia and loss of flesh and strength are apt to be in excess of the cough and expectoration. Examination of the chest discloses signs that indicate greater tendency to fibrosis and a more basic involvement than in tuberculous phthisis, while late in the course of the disease there is apt to be extension to the parapleural tissues or to distant parts, such as rarely, if ever, is observed in tuberculosis of the lungs.

The invasion is usually insidious and the early symptoms vary in accordance with the pathological variety of the affection. If the actinomycosis first attacks the pleura pain is likely to be the chief cause for complaint. There may be at the same time a gradual decrease in strength and the individual loses color and weight. He may have no cough or so little that it fails to attract attention.

A chest examination at this time will probably reveal signs of pleuritis, circumscribed or general, and with or without exudation. If fluid be demonstrated and an exploratory puncture be made the needle may, if it enter a pocket, reveal the presence of yellow or yellowish-white pus containing the characteristic granules.

In some cases the condition that arrests attention is an elastic subintegumentary phlegmonous mass which may resemble a semisolid malignant tumor or, from the appearance of the skin and the existence of one or more points of fluctuation, is plainly an abscess. On incision or aspiration of this swelling a yellowish, possibly foul-smelling, puriform material is obtained which consists of degenerated cells and the peculiar actinomycotic masses.

The case reported by Dr. E. Fletcher Ingals is one in point. The patient was an Italian laborer who was admitted to the Presbyterian Hospital because of a painful swelling on the right back in the interscapular region. The history was negative so far as its ætiology was concerned. The man had been working on a railroad and had slept on straw obtained from a neighboring farm. One night, after a trip to an adjacent city, he had felt ill with pain in the back and had been obliged to give up his job.

On admission to the hospital it was found he had a swelling about 8 cm. in diameter in the right interscapular space extending from the spine to the inner border of the scapula and from the third rib above to the seventh rib below. This tumor was raised in its highest part about 2 cm. above the surrounding niveau and the skin about the swelling was firmly attached to the underlying tissue. There was very little motion possible in consequence. Over the center of the swelling fluctuation could be detected, and there the skin was loose and movable.

Dr. C. Volini brought this patient to the hospital and placed him under the care of Dr. Emanuel Senn, who made an incision over the swelling, from which about a teacupful of grayish pus and blood-clots escaped. A counter opening was also made about two inches below. Drainage was established and healing by granulation encouraged. In the pus were many small yellow granules about the size of a pin's point, which, upon microscopic examination, proved to be actinomycosis fungi.

When Dr. Ingal's attention was directed to the case it was not certain whether the abscess communicated with the pleural sac or not, but Dr. Senn was inclined to think it did. The only departure from the norm found by Dr. Ingals in the chest was an area of moderate dullness on the right back extending from about 2.5 cm. above the level of the scapular spine to the inferior angle of this bone, and from the inner border of the vertebral column to 2.5 cm. outside of the inner margin of the shoulder blade. Over this dull area pectoral fremitus was diminished, vocal resonance was seemingly normal, breath-sounds were indistinct but no râles were audible. In the drain from the wound was a single yellow granule about 2 mm. in diameter which, under the microscope, was found to contain typical actinomycetes.

Dr. Ingals inclines to the view that in this case the infection occurred through a scratch of the skin made by the straw on which the man slept. From the well-known pathology and ætiology of actinomycotic disease it seems to me more likely that the abscess was the outward manifestation of a deeply seated process either in the pleural sac or, more probably, in the mediastinum posticum. Such a view would readily explain the absence of cough.

In the *broncho-pneumonic* form of primary actinomycosis of the lung the history is quite different. The early stage is marked by slight cough and scanty muco-purulent expectoration which may or may not contain the actinomycotic granules. Decline in strength and weight occur and the individual grows anæmic, often out of proportion to the pulmonary findings. These, as a matter of fact, may be very few and indefinite, possibly some harsh breathing and catarrhal râles but no dullness. This *bronchitic stage*, as it is called (Israel), merges after a lapse of months into one in which obvious pulmonary change is demonstrable. Nevertheless, Canalis has reported the case of a fifteen-year-old boy who manifested actinomyces in his muco-purulent sputa for a number of years without, however, definite changes in the pulmonary parenchyma or pleura. Inhalations of turpentine afforded some relief but did not cause a disappearance of the fungus from the expectoration
Fraenkel is inclined to doubt, and in this doubt I must con
process could remain indefinitely confined to the mucous mem
bronchi. The tendency of the organism to attack all structures
is too plainly proven. This, notwithstanding Chiari's observati
in which the fungus remained limited to the mucous lining of

With encroachment of the necrotic process upon the peribron
alveolar walls, etc., the cough is apt to increase and the expe

grow somewhat more profuse. The cough, however, is rarely exce though Lenhartz states that in the more advanced stage it may sc be very troublesome, especially at night.

The *sputum* is muco-purulent and contains blood. The quantity is not sufficient to constitute an outspoken hæmorrhage, differing respect from pulmonary tuberculosis. It imparts to the sputa a raspb color and herein is not unlike that of malignant disease of the lung sionally the expectoration may be offensive, distinctly yellow and qu ous. This is a great exception, however.

The patient now presents considerable cachexia, being thin, pale a A chest examination at this time will reveal more or less pronour extensive dullness over the lower part of one lung with evident r of the thoracic wall. Breathing is bronchial, but râles are few, if p all, and signs of excavation are very indefinite.

These findings are usually confined to one side but it is stated very chronic cases both lungs may become ultimately affected.

The case in the care of Dr. Billings is one of this kind. The was a cigarmaker, aged twenty-one years, who had a sudden onse days before admission, which was characterized by chill, cough, f slight muco-purulent expectoration. Shortly after admittance and a severe coughing fit he brought up a large mass which, on exam proved to consist mainly of tobacco leaf inclosed in purulent mate containing many of the sulphur-yellow, pinhead-sized granules chara of actinomycosis. The temperature then dropped to below 100° F. a remained for several weeks. The physical signs on admission we nounced dullness with suppressed breathing and many large and sma râles in a small area below the left clavicle, and in another area abo inches in diameter in the lower part of the left axillary region. T side was not involved. On treatment with iodide and X-ray expos râles disappeared, except an occasional large click in the infrac region.

The last stage of the disease can be said to have been reached v necrosis of tissue shows itself by swelling or abscess of the thoracic In all probability the fungus has penetrated into the mediastinal, su or other structures and has invaded the liver or spleen. Th continued suppuration, moreover, is likely to have induced amyloid eration of kidneys or other viscera. The case reported by Ascho Fraenkel's ward illustrates the features of this stage so well as to ju introduction here.

"A thirty-year-old laundress, after slight cough and greenish ex tion for a few months, suddenly became ill in December, 1892, w in the right side of the chest. At the date of her admission to the eleven months subsequently there was found a moderate pleuritic on the right side together with flattening of the upper front of the same side. In addition there was a loud systolic and diastolic murmu third right interspace close to the sternum as well as on that bon expectoration which was muco-purulent and free from tubercle bac fungi was frequently bloody. On November 15th aspiration of the right chest resulted in the removal of clear serum. Toward the end of December there appeared in the right inframammary region a swelling which extended to the lower costal arch, increased in extent and ultimately became fluctuating. Puncture of this abscess on January 8th removed a thick, foul-smelling pus which contained actinomycetes. At about the same time a fistula broke through the chest-wall at a point higher up and through this opening the same kind of pus was discharged. In spite of extensive incision of the abscess numerous fistulæ and small abscesses continued to develop. A typical actinomyces was demonstrated in the contents of a decayed tooth. The chest, especially at the right back, continued to retract and death took place March 22, 1894.

"Autopsy. Right lung. Adhesions bound the upper lobe on all sides to the pleura and in the lower inferior portion of the pleural cavity was about half a liter of pus. The internal wall of this empyemic zone was composed of disintegrated tissue having the appearance of cinder while the thickened pleura in front was permeated with irregular pus cavities. The whole middle lobe was converted into a grayish-green airless tissue which in places looked of gelatinous transparency and in others contained bright-yellow softened areas. The ascending aorta was dilated and firmly united to the fibrous middle lobe and to the sternum."

Physical Signs.—From what has been said concerning the symptomatology of pulmonary actinomycosis it is apparent that there are no distinctive findings on the part of the lungs. In the bronchitic form the picture presented is merely that of a chronic bronchial catarrh and as such needs no description here.

Inspection.—In fortunate cases there may be an external tumor or a discharging abscess, in the pus of which may be found the characteristic yellowish granules. When no such external manifestations are present, the chest will probably display no changes of moment. Occasionally, however, there may be some retraction of the affected side.

Palpation.—Pectoral fremitus may or may not be altered. In the broncho-pneumonic variety this may be exaggerated while in the pleuro-pneumonic type, vocal fremitus is likely to be diminished or absent. Such indeed is likely to be the palpatory finding in most cases rather than the reverse, i. e., an increase of fremitus.

Percussion.—There will probably be more or less dullness according to the character and extent of the pulmonary and pleural process. When loss of pulmonary resonance is discovered, it is usually basic. It is possible, however, for the dullness to be apical. In extent this change in the percussion-note is likely to be circumscribed. When the dullness discovered
apex it is not likely to possess the configuration seen in tuberc
is, to spread down the back to a greater distance than in front.

Auscultation.—The breath-sounds are likely to be bronchial
vesicular over the areas of impaired resonance. In other parts
have a harsh or indeterminate character. Catarrhal râles may
bronchitis coexists but for the most part they are likely to b

vague. The voice-sounds may be bronchophonic in the broncho-pneumonic and diminished in the pleuro-pneumonic variety. Although small cavities may be formed, they are not apt to reveal their presence by the auscultatory signs of vomica. The absence of more definite changes in the respiratory murmur and of adventitious sounds is probably owing to the tendency of the disease to burrow its way along the connective tissue.

It will be found by perusal of the other forms of fungus disease of the lungs, that like them actinomycosis presents no exception to those modifications of the breath-sounds which are always found whenever the parenchyma undergoes consolidation and the pleura becomes thickened. The main point to be borne in mind is the tendency of this disease to occupy circumscribed areas.

Diagnosis.—The recognition of actinomycosis of the lungs will rarely if ever be possible without the detection of the fungus in the sputum or in the fluid obtained from the pleural cavity or an abscess. The not unreasonable assumption that this disease exists much more frequently than it is recognized indicates the wisdom of not confining sputum examinations to a search for tubercle bacilli. The possibility of its presence should be kept in mind whenever one is called on to pronounce an opinion on the nature of obscure lung affections occurring in persons exposed by occupation or environment to this sort of infection. Whereas a definite history of exposure may be of positive value in some cases, still equal weight cannot be attached to a negative anamnesis, as shown by Ingal's and Butterfield's cases.

As long as the disease is in its bronchitic stage or is confined to the lungs and their investing membranes without having perforated the chest-wall one can scarcely hope to do more than make an inferential diagnosis from the clinical history and physical signs because of the lack of characteristics that distinguish it from certain other more frequent disorders.

The following points may be borne in mind, however: (1) the tendency to anæmia and debility out of relation to the physical signs; (2) the tendency of actinomycosis to affect the lower rather than the upper portions of the lungs, although the exceptional localization at an apex must not be forgotten; (3) the tendency to pleural thickenings and to extensive fibrous contraction of the lung; (4) its tendency to tumefaction and abscess formation in the parietes of the chest.

Differential diagnosis must be made between actinomycosis and the following disorders:

(*a*) Tuberculosis of the lungs. If sputum examination does not at once settle the nature of the affection, help will be found in the apical localization, the greater disposition to demonstrable softening and cavity formation, its possibility of pronounced pulmonary hæmorrhage and its liability to earlier and more frequent involvement of both lungs.

(*b*) Malignant disease of the lungs. Here the main points are a possible history of antecedent or coexisting cancer of some other part, the likelihood of pressure-phenomena and dyspnœa, the absence in the majority of cases of fever and symptoms of pus-infection, negativeness of sputum examinations, and the infrequency of localized tumefaction on the chest-wall. Should examination of the expectoration or pleural fluid yield results it will at once determine the diagnosis.

(*c*) Syphilis of the lungs. This disease possesses some features similar to those of actinomycosis, especially its tendency to localization in the middle or basic parts of the lung. The difference must be found in the negativeness of sputum examinations, the history of specific infection or evidence of luetic stigmata on skin or in other organs, the absence of febrile and hectic phenomena and of abscess formation. Should a gumma of the thoracic parietes exist its aspiration will probably differentiate it from actinomycosis.

(*d*) Echinococcus cyst of the lungs. In this disease the points of difference are mainly the possible detection of the bladders or hooklets in sputa or fluid withdrawn by aspiration, the tendency in case of large cysts to bulging of the chest-wall and pressure on adjacent organs rather than to shrinkage, the greater liability to serious hæmoptyses and the less pronounced anæmia and weakness in the earlier stages of the disease. For additional data the reader is referred to the appropriate chapter.

Prognosis.—This may be said to be ultimately hopeless unless the disease be so circumscribed and otherwise fortunately circumstanced as to admit of surgical interference which, however, is a remote possibility. The duration of the disease may be put down as from months to one or two years. Healing of an external abscess may follow spontaneous rupture or surgical treatment and arouse hope of cure, but the tendency of the disease to creep onward will be ultimately demonstrated.

II. ASPERGILLOSIS

This disease which furnishes another example of mycotic destruction of the lungs has been extensively studied in France and Germany where numerous cases have been reported (Virchow, Friedreich, von Dusch, Pagenstecher, Fürbringer, Kohn, Weichselbaum, Sticker, Widal, Renon, Dieulafoy Chantemesse). The views first entertained were those advanced by Virchow, namely, that the affection could not be regarded as a primary one but that the infection with the aspergillus was secondary and hence that the destructive changes observed were referable to the disease on which the mycosis was engrafted. The researches of Renon, Kohn and others have proven conclusively, however, that although Virchow's views may obtain in some instances, they do not in all.

Ætiology.—The disease in question is caused by a fungu
gillus fumigatus, which is the mold commonly seen on grain
in Fig. 83, it consists of a mass of fine transparent branched
are joined together and constitute the mycelium. These threa
the surface and in the substance of the object on which it
straight filaments are attached to the mycelium and on the su
are radiating protuberances which support a chain of spores.

This fungus gains access to the body either by being inhaled or by being carried into the mouth along with the grain or meal on which it feeds. Hence this form of pulmonary mycosis is observed most frequently among persons whose vocations bring them into direct contact with the aspergillus.

In France the disease is seen chiefly among feeders of pigeons, hair-combers, millers. The pigeon feeders are said to take the grain into their mouths and then force it into the throats of the birds. The hair-combers use flour in connection with their work and hence are liable to inhalation of the fungus along with the fine dust of the flour. Fowler makes the interesting statement that household pets are unable to live in the homes of these hair-combers, birds dying in from two to three weeks, dogs in three months; cats only are insusceptible.

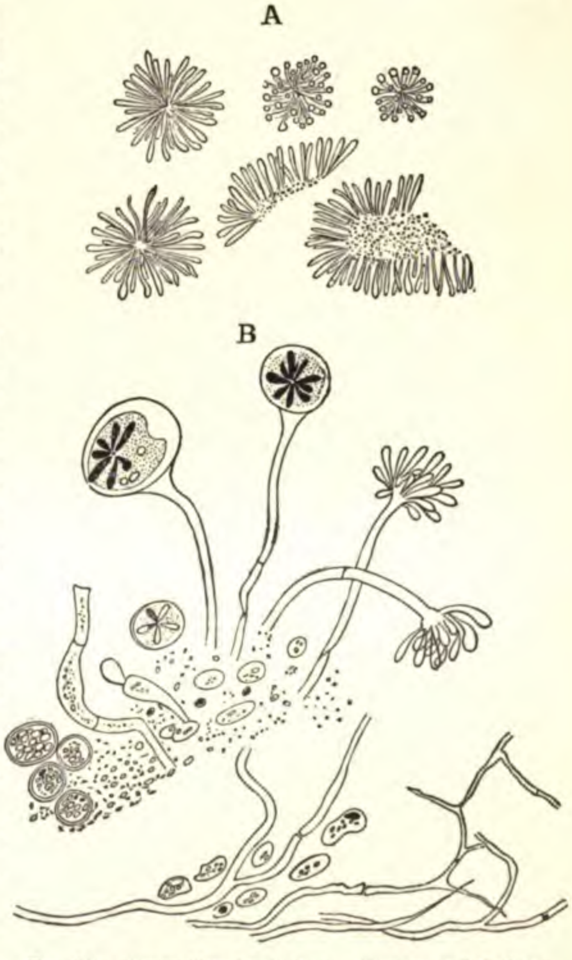


Fig. 83.—Aspergillus fumigatus. (Fowler and Godlee, published by Longmans, Green & Co.)

Morbid Anatomy.—The changes produced by the aspergillus fumigatus in the lungs are of a chronic nature, being inflammatory, destructive and productive. The areas are not extensive and are at first catarrhal with infiltration and exudation of cellular elements characteristic of broncho-pneumonia. These foci of inflammation tend to necrosis, and ultimately show the characters of gangrene though without the extreme putrescence of ordinary gangrene. On cross section such an area may look not unlike a sponge or honeycomb. The small cavities on which this appearance depends may be true vomicæ or dilated alveoli (West) in which the aspergillus may be discovered. In the course of time, proliferative and reparative changes may occur and, the gangrenous tissue being cast off, the disease may be brought to an arrest.

Symptoms.—The disease presents no characteristics that mark it from other destructive lesions of the lungs and, in its earlier stages at least, it may be mistaken for pulmonary tuberculosis. There is more or less cough with little or no expectoration, such as there is being of a catarrhal nature. In some instances hæmoptysis is said to occur, but as aspergillosis is not infrequently engrafted upon tuberculosis it may be that the hæmorrhages are due to this latter disease.

Strength is lost gradually and the patients lose appetite and ability for work. They may have some pyrexia and in general present a clinical history sufficiently like that of tuberculous disease to account for the readiness with which it may be mistaken for ordinary phthisis.

Examination of the lungs is likely to discover an area of dullness at one apex with bronchial breathing and possibly a few râles. If the sputum be carefully examined it may be found to present a brownish or grayish-green appearance, to contain the aspergillus fumigatus within the bloody purulent masses or possibly in fragments of disintegrated lung tissue.

The disease may progress under ever-increasing cachexia to death, or arrest may take place, the expectoration gradually losing the fungi and the general symptoms becoming gradually less.

Physical Signs.—These are not characteristic and, being those of induration and destruction of lung, do not need detailed description.

Inspection and palpation are negative excepting in so far as the former detects evidence of wasting and anæmia, and the latter, possible increase of tactile fremitus over the affected portion of the lung.

Percussion will disclose more or less pronounced and extensive loss of pulmonary resonance, possibly of tympanitic quality, depending upon the extent of the changes.

Auscultation.—The breath-sounds are likely to be altered in accordance with the stage of the process, being harsh in the early stage and bronchovesicular or bronchial after the disease has led to destruction and fibrosis. Râles may or may not be present. Signs of cavity are either wanting or indistinct.

Diagnosis.—This is only possible by the discovery in the sputum of the mycelial threads or of the whitish or yellowish-white bodies constituting the fruit. In any case of pulmonary disease resembling tuberculosis, the expectoration should be examined not alone for bacilli but for this and other forms of fungi. If the sputum be stained with a weak watery solution of saphranin or thionin (Renon), the fungus if present will be easily detected. Only by proper cultural and experimental procedures can the exact nature and pathogenic properties of the threads found in the sputa be proven.

Without microscopic inspection of the fresh sputa the diagnosis
be conjectural and then must be based on the history of exposure
tion through the occupation, rather than on physical signs.

Prognosis.—The outcome of the disease must always be dou
covery is not impossible but must depend on sloughing off an
of the necrotic focus, at best a somewhat doubtful continge

III. STREPTOTHRICOSIS

This is an affection produced by the streptothrix which is a v
organism intermediate between molds and bacteria. The rarity
form of pulmonary disease may be judged of from the fact that in
paper by Ashton and Norris, Journal of the American Medical Ass
September 9, 1905, these authors were able to cite but 26 authent
of pulmonary streptothricosis. Of these only 7, including their ow
published in the United States. For a summary of these collected i
the reader is referred to the paper just mentioned.

Ætiology. *The Organism.*—There seems to be some doubt co
the proper classification of the streptothrix. It forms a rather ill

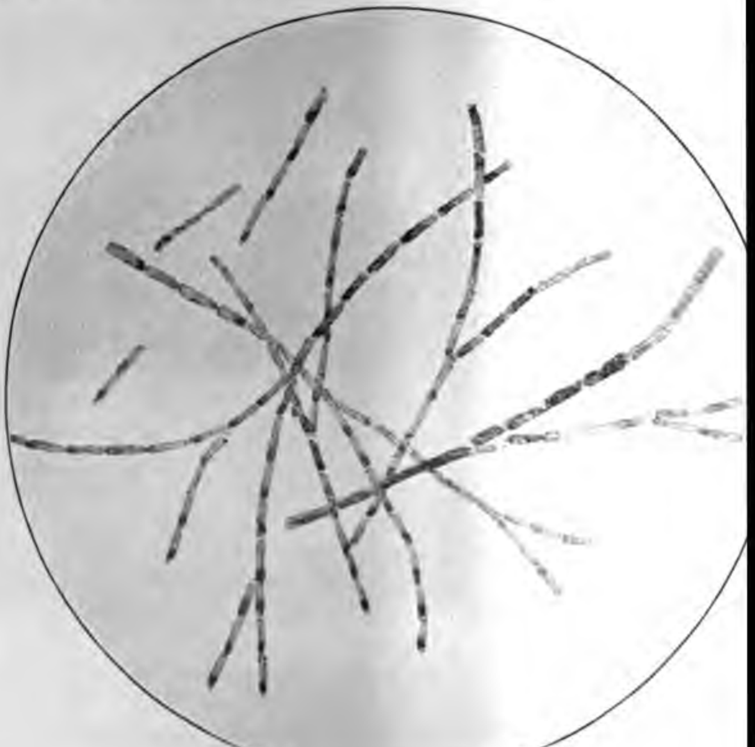


Fig. 84.—Streptothrix.

genus of fungi or molds which, while closely resembling bacteria,
sesses some features similar to those of the hyphomycetes. Acco
Flexner the organism in question develops from spores into branching
having a dichotomous arrangement. These threads alternately gr
colonies whose appearance is that of a mass of radiating filaments or

(Fig. 84). Certain of the threads become fruit hyphæ and these break up into chains of round spore-like bodies.

The streptothrix is not readily stained with carbolfuchsin, but reacts better to the Weigert-Gram method. Even then it is said by Fraenkel to stain poorly and irregularly so as to resemble cocci. Seen under the microscope the organism appears as long, fine, homogeneous, glistening threads which occur in clumps and exhibit true branching arrangement. It may occur alone in the sputum of individuals afflicted with streptothricosis but is not infrequently associated with other micro-organisms, especially streptococci. It is on this account that doubt has sometimes arisen as to the real nature of some cases in which the streptothrix has been discovered.

Predisposing Conditions.—Nothing is known concerning the factors that promote infection of the respiratory organs by this organism. The age in 16 cases in which this was definitely stated ranged from twelve to seventy years, the majority ranging between thirty-six and fifty-six. Of the 17 cases in which the sex was given, 12 belonged to the male and 5 to the female sex, a preponderance on the part of men not explicable unless possibly by the nature of their work.

The occupation of the patient was definitely stated in 14 cases and showed considerable diversity. Thus, there were 4 laborers, 4 women whose vocation was put down as housework, and 1 each as follows: glass cutter, steel molder, carpenter, business man, soldier, farmer, school child, and brickmaker. From this list it is apparent that in occupation as such there is no factor which can be laid hold of as definitely predisposing to the disease, unless possibly it be the inhalation of dust impregnated with the organism.

Inhalation.—Inasmuch as we possess no data pointing conclusively to the mode of entrance of streptothrix into the lungs, and since the reported cases seem to furnish *prima facie* evidence of having been primary in these organs, we are probably justified in the assumption that the organism gained entrance by inhalation, the same as in other forms of fungous disease of the lungs. Thus Mayer is said to have observed cases of pulmonary streptothricosis in Australasia, which developed soon after a sandstorm.

Morbid Anatomy.—The affection under consideration may appear in either of two forms, acute or chronic. In the former, which is by far the more common, the gross appearances bear considerable resemblance to changes seen in pulmonary tuberculosis. There are caseous or necrotic areas which are in reality foci of broncho-pneumonia that have undergone rapid softening. Surrounding these necrotic areas the tissue is found to be in a state of softened consistency in consequence, as the microscope shows, of infiltration with round cells. Where abscess cavities have been formed their walls present a ragged, shreddy appearance quite unlike the vomicæ found in tuberculosis (Fraenkel). The streptothrix has been demonstrated in the purulent material of such foci as well as in the pleural cavity (Mayer).

The chronic form is represented by a case reported by S. Flexner in 1898. The lungs were voluminous and free from adhesions. The left upper lobe was consolidated throughout save along its anterior margin. Where this was most firm, as at the apex, the tissue presented a gray, opaque appearance.

In some areas there was beginning softening "disintegration with cavity formation." In the lower portion were broncho-pneumonic foci and the intervening lung substance was soft and œdematous. In the lower lobe were partly calcified nodules of the size of a split pea which resembled tubercles. The right lung contained similar nodules as well as foci of caseation of which one was in a state of softening. Reactive encapsulation of such caseous areas was absent.

In addition to the changes in the lungs most of the recorded cases have shown metastatic foci in other organs, as brain, liver, heart, spleen (Engelhardt and Loehlein), in the skin (Scheele and Petruschky), peritoneum and omentum (Flexner), kidneys, bladder, spinal cord, bronchial lymph nodes, etc.

Symptoms.—The clinical picture of pulmonary streptothricosis is very similar to that of tuberculous disease of the lungs. In the acute variety there are symptoms of more or less rapid destruction of pulmonary tissue, cough and profuse expectoration consisting of muco-purulent or purulent material having a yellowish or greenish-yellow color and possibly containing, as in one of Mayer's cases, elastic tissue, blood and alveolar cells. There is pyrexia which, when metastases occur in other parts, may be like that of pyæmia (Ashton and Norris). These cases go on to a rapidly fatal termination.

The disease is apt to set in abruptly with severe cough, bloody sputum and pain in the side as in the case reported by Buchholtz in 1897, and physical examination discloses signs of apex disease, dullness and numerous râles, while in one or other of the pleural cavities may be an exudate of either serum or pus. In Buchholtz's case the effusion was serous at first and scanty, but subsequently became purulent and contained streptococci. In other instances the signs on the part of the pleura may point to obliteration of the sac by adhesions as shown by a negative exploratory puncture. Such was the result in one of the cases reported by Ashton and Norris.

Buchholtz's patient manifested a temperature of the inverse type which, in the morning hours, ranged between 38° and 39° C. (100.4° and 102.2° F.). His strength failed steadily, emaciation progressed and death took place at the end of eight weeks from the beginning of his illness. At the autopsy the left lower lobe was adherent to the diaphragm and contained a firm area of broncho-pneumonia. This area was the size of a hen's egg, and was in a state of necrosis. In the corresponding lobe of the right lung were two large, communicating abscess cavities with ragged walls, which cavities had broken into the pleural sac. A smear of the pus showed abundant streptothrix masses.

One of Ashton and Norris's cases recently reported was that of a twenty-two-year-old mulatto who was first admitted to the surgical ward of the Philadelphia General Hospital on account of inguinal buboes. After these had been appropriately treated he was transferred to a medical ward because of symptoms indicative of some pulmonary complaint. His symptoms consisted of persistent cough, with copious fœtid expectoration and pain in the left side and back.

Examination showed pulse 90, respirations 35, temperature 102° F. The left side of the chest displayed marked dullness extending from the base up to the seventh rib and impairment of resonance at the apex anteriorly, together with a few subcrepitant râles. Posteriorly the apex was dull with broncho-vesicular expiration and great numbers of mixed râles. Over the base of this and also of the right lung there were in addition to dullness, feebleness of the breath-sounds and diminution of vocal fremitus. Over an area 2½ inches in diameter beneath the lower third of the left scapular region were amphoric respiration, whispering pectoriloquy and moist resonating râles. The left half of the chest was sensitive to percussion and respiratory movements were restricted. Heart and other organs were negative, save that the urine was scanty and showed a trace of albumin and a few hyalin casts. The leucocytes ranged from 9,000 to 13,600 and were made up chiefly of the polymorphonuclear variety.

The course of the disease was characterized by emaciation, profuse night sweats, persistent cough, large amounts of purulent sputum and several severe hæmoptyses. In a word the clinical picture was that of pulmonary tuberculosis of an acute type. Sixteen different examinations of the sputa for tubercle bacilli were invariably negative, but at length streptothrix was reported present, associated with other bacteria in great variety. On March 29th, more than six months after admission, it was noted that the clubbing of the fingers observed at entrance had greatly increased.

The resemblance of this case to the features of pulmonary tuberculosis was so marked, that the diagnosis of that disorder was entered. When, however, the streptothrix was discovered in the sputum the true nature of the affection was recognized and the diagnosis was changed accordingly. In spite of the ordinary medicinal treatment resorted to in tuberculous cases, forced feeding with milk, eggs, etc., and open air taken on the fire escape, the patient's symptoms had not materially improved by the fore part of April. He declared, nevertheless, that he felt strong enough to return to work and insisted upon leaving the hospital. He left, accordingly, and was lost to further observation.

The second case reported by Ashton and Norris was that of an Italian, aged thirty-six, who was admitted to the same hospital March 23, 1905, complaining of symptoms pointing to a left-sided pleuritis. The family and personal history was entirely negative. The present illness began one month prior to admission with cough which steadily increased with lapse of time, and was soon followed by muco-purulent expectoration, loss of weight, night sweats, anorexia and several attacks of hæmoptysis.

The man was well developed and fairly well nourished, showed a well-formed chest, marked clubbing of the fingers, marked accentuat
aortic second tone, a palpable spleen, normal reflexes, diminish
and painful respiratory movements on the left side of the chest.
existed over the lower two thirds of this side, with feeble, dist
sounds, and absent vocal fremitus. At the apex were impaired
increased fremitus and cogwheel inspiration with broncho-vesicul
tion. Similar changes were present in the right upper lobe

râles were heard from time to time throughout both lungs, but chiefly in the left.

March 26th, it was noted that the symptoms consisted of pain in the left side, incessant cough and copious, thin, muco-purulent expectoration. There were fever of an irregular type, night sweats and, in short, such symptoms as seemed plainly to indicate pulmonary tuberculosis. The sputa, however, did not contain tubercle bacilli and at length streptothrix was demonstrated which settled the diagnosis.

By May 9th the patient's dyspnœa had increased, his emaciation was more apparent and his fingers were more clubbed. The expectoration remained of the same character as previously, save that at times it was almost aqueous and often amounted to over a quart in twenty-four hours. This was taken to point strongly to the presence of bronchiectasis, a condition which according to the reporters has occasionally been found. Soon thereafter the patient presented an almost cachectic appearance and looked very ill. The urine was negative, excepting that it was small in quantity. The blood showed a moderate reduction in the red cells and 7,400 leucocytes, of which the polymorphonuclear cells constituted 77 per cent and lymphocytes 13.5 per cent.

In the *chronic* form of this disease the symptoms also bear a close resemblance to the features of pulmonary tuberculosis. They are not so severe as in the acute or subacute cases of streptothricosis just depicted. Flexner's patient was a negro of seventy who was admitted to Johns Hopkins Hospital, Baltimore, on account of chronic cough and other symptoms suggestive of tuberculous disease of the lungs. Expectoration was absent, and hence the nature of his affection had to be determined as far as possible by the chest examination. The results of physical exploration of the lungs is not specifically stated by Flexner in his report; but it may be inferred that there were dullness and bronchial breathing over the left upper lobe.

Physical Signs.—These are very like those found in other pulmonary complaints which lead to consolidation, disintegration and cavity formation either with or without pleuritis. They do not, therefore, present anything that may be considered characteristic.

Inspection.—The chief change that may be noted by the eye, is restricted and delayed or possibly abolished respiratory movement over one or the other side, in consequence of the pleuritis so often present. There should also be retraction and diminution of motion at the affected apex.

Palpation.—Pectoral fremitus is decreased or absent over the base in case there is either an exudative or adhesive pleurisy, while at the apex vocal vibrations are increased. Such were the findings in all of the cases which have been briefly described in this chapter.

Percussion.—This detects more or less dullness, the intensity and location of which are determined by the changes produced in the lungs. If pleurisy is the predominant condition noted, the percussion findings will be more marked at the base as in Buchholtz's case which showed flatness at the base of the right lung in consequence of a purulent exudate. In all cases there has been found more or less pronounced impairment of resonance at the apex of either one or both lungs. In some instances there may be very little normal pulmonary resonance on either side.

Auscultation.—The changes detected on auscultation are various. At the base over the seat of the usually present pleuritis, breath-sounds are feeble or absent, while at the apex the consolidation leads to broncho-vesicular rather than to distinct and typical bronchial respiration. Moist râles of a subcrepitant or mixed character are apt to be heard chiefly over the upper portion of the lungs, but in number and situation they depend upon the extent and location of the necrotic areas. Distinct signs of cavity are not always observed either because the abscess cavities are too small or are concealed by the signs of pleuritic adhesions or exudate.

Diagnosis.—This can be determined only by the discovery of streptothrix in the expectoration or in the pus obtained from any metastatic abscess that may chance to coexist with the signs and symptoms of a caseating pulmonary affection. By physical signs alone it is doubtful if a correct diagnosis may ever be possible.

Prognosis.—This is anything but encouraging. As far as I am able to learn all of the cases of pulmonary streptothricosis thus far reported have terminated fatally. Indeed, when the nature of the changes caused by the organism in the lungs is considered, it is difficult to see how any other than a fatal issue is possible.

IV. BLASTOMYCOSIS

Much interest has been aroused in the last few years by reports of cases of generalized blastomycosis, under which term are signified certain lesions due to the action of the fungus blastomycetes. Among the lesions which are produced the most conspicuous are those of the skin, and hence the cases that have been reported have been especially observed and studied by dermatologists. When this disease is generalized the internal organs are involved and among these the lungs. On this account it is necessary to devote a few pages to the consideration of this newly discovered form of pulmonary disorders.

The first instance recorded was that of Busse and Buschke, which was published in Virchow's *Archiv* in 1895. There were cutaneous, subcutaneous, visceral and bone lesions, and the patient succumbed in from thirteen to eighteen months after having come under observation. There is some doubt as to the situation of the lesion first observed, but it appears to have been on the tibia and thence to have spread to the knee joint.

The second case reported was that of Walker and Montgomery published in the *Journal of the American Medical Association*, April 5, 1902. Systemic involvement did not ensue until cutaneous lesions had existed for several years. The skin lesions were curetted and cauterized, but nine days subsequently elevation of temperature occurred, the pulse-rate became increased, respirations were rapid, emaciation and dyspnœa set in and the case terminated fatally in forty-three days after the operation. At the autopsy the lungs as well as other viscera were found involved, but such were the characters of the changes that the patient was believed to have died of acute miliary tuberculosis. When, later, the attention of Drs. Walker and Montgomery was called to the subject of blastomycosis they recollected their case, reexamined their slides and became convinced of its having been one of this disease.

The third case was that of Ormsby and Miller reported in the *Journal of Cutaneous Diseases*, March, 1903. It is particularly worthy of note that in this one the initial skin lesion did not appear until two months after the development of certain lung symptoms, namely, cough, blood-streaked sputum and weakness which were by the patient attributed to having taken cold.

Temporary improvement in the general condition occurred, but the patient then grew pale, emaciated, anæmic with loss of appetite, a coated tongue and foul breath. The urine was negative, but temperature was elevated, pulse accelerated and respirations more frequent than normal. Large numbers of subcutaneous nodules and abscesses as well as cutaneous ulcers occurred. The lungs on examination showed bronchial breathing and other evidences of disease. Laryngeal ulceration was noted and the case was consequently examined with care for evidence of tuberculosis. Bacilli were absent in the sputa, however, and there was no reaction to tuberculin. Smears from pus taken from various organs and tissues after death, animal inoculation and cultures were all negative as regards tuberculosis, but by cultures and otherwise it was plainly shown that the lesions were those of blastomycosis.

The fourth case was that of J. H. Cleary reported in the *Transactions of the Chicago Pathological Society*, May 9, 1904. It was found at the *post-mortem* examination to be one of generalized blastomycosis, but during the time the patient was in the Cook County Hospital the true nature of the disease was masked by certain special symptoms of cough, muco-purulent expectoration, loss of weight, anorexia, hoarseness and œdema of the lower extremities. The urine contained albumin, granular and hyalin casts. The body temperature was always subnormal and there were no cutaneous lesions. No tubercle bacilli were found in the sputa. At the autopsy the blastomycetes were found in the lungs, liver, spleen, adrenal bodies and myocardium. The changes in the lungs will be referred to again.

The fifth case published up to the time of this present writing is that of Eisendrath and Ormsby which appeared in the *Journal of the American Medical Association*, October 7, 1905. The patient whose case is described in this report was still alive at the time the paper appeared, and was examined by me the latter part of September, 1905. Reference will be made to this case again under the symptoms of the affection.

A case reported by Ophüls and Moffitt in the *Philadelphia Medical Journal*, 1900, is classed as one of blastomycosis by some, but by others as one of protozoic disease. The organism closely resembled in its cultures the blastomycetes, but in the tissue it developed by spore formation and not by budding as is characteristic of the blastomycetes. Nevertheless, in its clinical and pathological findings this case is very similar to those that undoubtedly belong to the group of blastomycosis.

In addition to the foregoing published instances there have been observed several additional cases of generalized blastomycotic infection; so that at the present writing there have been something like nine instances of the disease observed in Chicago and its vicinity. It is probable that in the years to come many additional cases will come to light, and hence that this form of fungous disease will assume great importance.

Ætiology. *The Organism* (Fig. 85).—This is a fungus of the yeast variety and when properly stained with methylene blue and orange tannin according to Unna's method it appears as a round, oval or slightly irregular body having a well-defined, double-contoured, homogeneous capsule which resists the prolonged action of alkalies and acids. It has a finely or coarsely granular protoplasm which is usually separated from the capsule by a clear space of varying width. A clear vacuole, varying greatly in size, is seen in some of the organisms. The diameter varies as a rule from 7 to 20 microns, though both larger and smaller forms may be encountered. Budding forms in all stages of development have been noted.

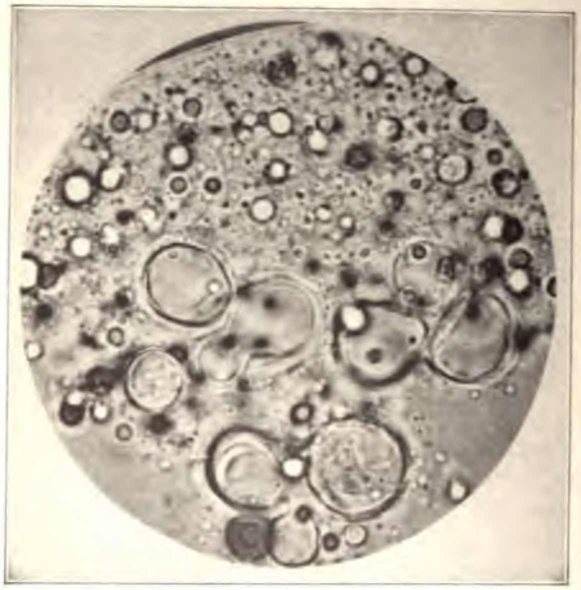


FIG. 85.—Blastomycetes in sputum. Note budding. (Eisendrath and Ormsby.)

The foregoing description is taken from F. H. Montgomery's paper on Cutaneous Blastomycosis which appeared in the *Journal of the American Medical Association*, June 7, 1902. From the same article is copied the following account of the methods of cultivating the fungus in question:

Cultures.—The organisms may be obtained from the miliary abscesses and also from the teased tissue and pus. The best method is to clean thoroughly with alcohol or ether a surface showing miliary abscesses. Some of the smallest and deep-seated abscesses are then opened with a disinfected needle, preferably with the aid of a magnifying glass, care being taken to avoid damaging the surrounding tissue. The best media are beer-wort, glycerine and glucose agars. These media are now to be inoculated with minute quantities of the tenacious mucus or muco-pus obtained from the abscesses and will in time show pure cultures of the organism; and Montgomery states that more than 50 per cent of his tubes showed pure cultures. It is considered well to inoculate as large a number of tubes as possible since it sometimes happens that the organism grows on one or two media and not on others.

Maltose agar (according to Sabouraud's formula) gives a more luxuriant growth with some of the fungi, but, on the whole, has been less successful, Montgomery says, in obtaining cultures from the abscesses than glycerine or glucose agar. It is said that a medium which is slightly acid is most sure of results. Pus cocci may contaminate the growths taken from teased tissue and pus, but pure cultures of the fungi have been repeatedly obtained from the pus of abscesses of considerable size, which fact is thought to indicate beyond doubt that the organism is in itself pyogenic. Fig. 86 taken from Eisendrath and Ormsby's paper shows the cultures well.

Organisms from different cases have varied so widely in their cultural features that it is considered quite possible that they will have to be classed in distinct botanic groups, and yet individual organisms have been found to differ greatly according to the media employed and with other conditions of culture. Montgomery thinks, as a result of his investigations, that in some cases there may be two varieties of a given species present, which circumstance might explain some of the observed phenomena and would accord with Hansen's findings who showed that in nature the yeast fungus often occurs in groups of allied varieties.

The time required for the development of the different organisms in the original cultures is said to vary from two to sixteen days, in the majority of instances from two to eight days. Subcultures appear in from two to five days.

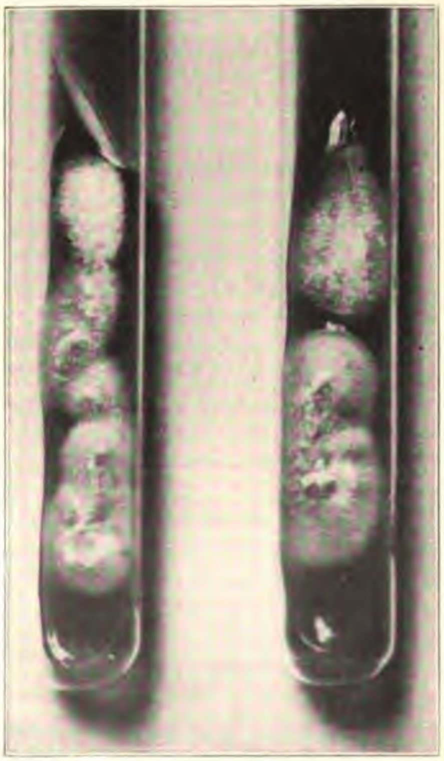


FIG. 86.—Blastomycetes in culture. (Eisendrath and Ormsby.)

Conditions Favoring Infection.—These are not known any further than that in a few cases of cutaneous blastomycosis there was a history of antecedent trauma. Neither is it known why some of the yeast fungi are pathogenic while others are innocuous. In the reported instances of generalized blastomycosis the involvement of the viscera seems in most cases to have been secondary to the development of the skin lesions.

The atrium of infection in three of the cases was probably the lungs. Thus in Cleary's case there were no cutaneous lesions whatever and the clinical picture was that of a pulmonary affection. In Eisendrath and Ormsby's case also the lungs were thought by the reporters to have been the first structures attacked. In Ormsby and Miller's case the clinical picture was that of a chronic lung disease, and hence the assumption seems warranted that in it also the port of entry for the organism may have been the lower respiratory tract.

When the skin is the primary seat of infection the lesions are of such a nature, I refer in particular to the subcutaneous miliary abscesses, that it is not difficult to account for visceral or systemic infection by way of the blood or lymph channels. When the lungs appear to be the primary point of invasion it is probable that the fungus is inhaled and finding the protection of the bronchial mucosa inadequate for some reason or other takes root and develops in the same manner as do other organisms. Unhygienic environment may furnish favorable conditions, but on this point we know nothing definite.

Morbid Anatomy.—The morbid anatomical changes found in the lungs are well described in the *post-mortem* reports of both Walker and Montgomery's case and of that by Cleary. Consequently, in the following description liberal use will be made of their accounts.

Upon removal of the lungs the attention is attracted by numerous whitish-gray or yellow nodules upon their external surface. These bodies are hard, project slightly above the surface and in macroscopic appearances resemble miliary tubercles. In size, however, they are larger, ranging from that of a pin's head to that of a pea. In places the pleura is adherent, while between the adhesions where the membrane is free from nodules its luster seems normal. The nodules are likely to be seen on both lungs but not in equal numbers.

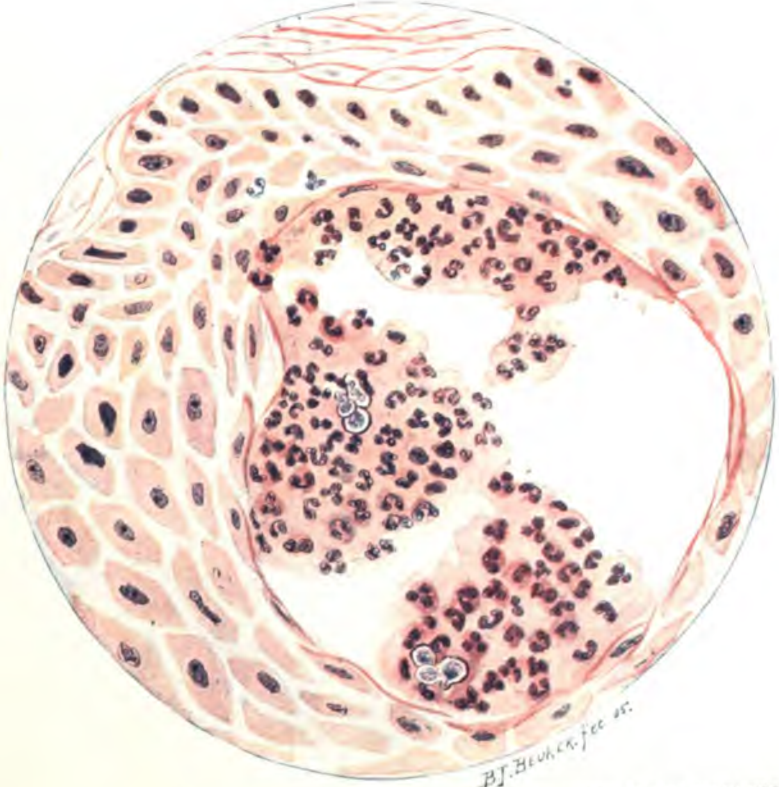
On section the nodules are found quite uniformly distributed throughout the organs, yet with a tendency to form clusters. Their line of demarcation is sharply defined and the intervening lung tissue looks unchanged. The situation of the nodules is in the perivascular and peribronchial connective tissue. They are found to have yellowish centers from which a scanty amount of pus may be squeezed. Furthermore, on cross section of the bronchi these are found to contain a muco-purulent material.

Microscopically (Plate IX) the miliary masses are seen to be made up by aggregations of polymorphonuclear leucocytes having a clusterlike arrangement that seems to indicate a coalescence of several foci. About such conglomerate masses mononuclear leucocytes chiefly abound and these latter cells are also found between the aggregations of the polymorphonuclear variety.

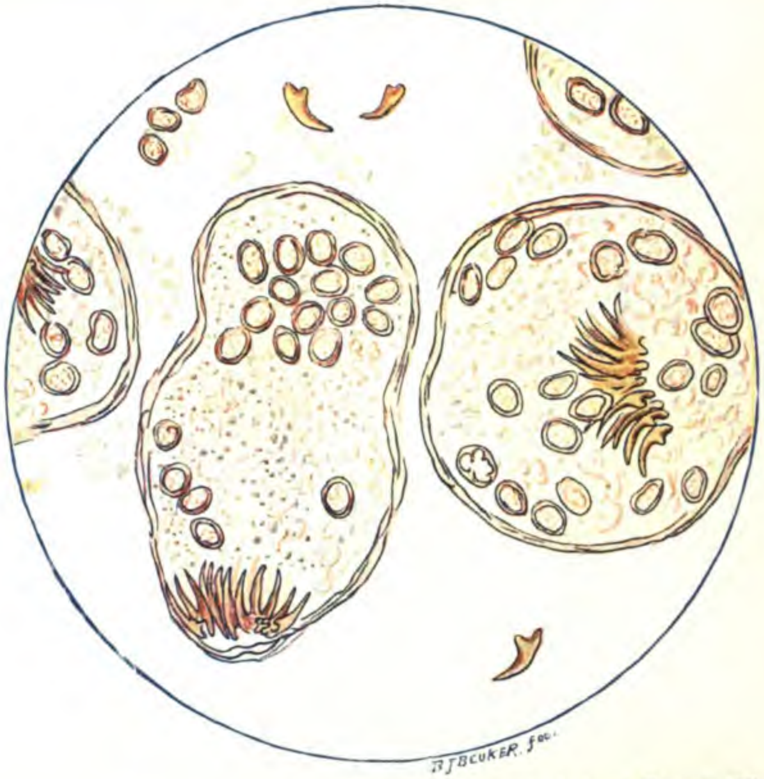
Fungi of varying size are also seen in the nodules, being for the most part in the peripherally located cells. The alveolar structure of the intervening pulmonary tissue may be normal, but the alveoli are apt to be filled with a granular material composed of mononuclear leucocytes and masses resembling fibrin. In most of the alveoli a commencing growth of connective tissue may be recognized.

The average diameter of the nodules is said to be from 2 to 4 mm. (Cleary); in many the central necrosis is a marked feature. Many large

PLATE IX



A.—Miliary Abscess in Blastomycosis Cutis, Showing Characteristic Organisms. (Stained with Hæmatoxylin and Eosin.)



B.—Small Cysts and Hooklets from Sputum in Case of Pulmonary Echinococcus Disease. (Unstained.)

giant cells occur and in some of them as many as six or eight organisms may be seen. Wherever bronchioles are found in these blastomycotic areas they are occluded by leucocytes and in some of them the fungi are very numerous. Although the organisms are found in the periphery of the nodules they are rarely to be seen in the necrotic centers.

Loeb in his histological report in Walker and Montgomery's case states that changes may occur in the organism itself. It usually has a darkly stained center from taking up the nuclear stains. In the peripheral portion of some specimens stained with methylene-blue many fine blue granules may be seen. In some of the organisms the central body takes up the stain only slightly or not at all. Budding of the organisms may occasionally be recognized while other specimens may be seen having a central round body of a mulberry-like appearance, the segments being of equal size.

Loeb concluded that the blastomycetes do not cause an epithelial proliferation in the lungs similar to the lesion found in the skin, but changes of an inflammatory character similar to those found in pulmonary tuberculosis.

Other changes may be found in the skin, consisting of purplish verrucose lesions elevated above the surrounding tissue. These raised areas are soft, friable, spongy, and droplets of pus may be expressed from beneath them. The skin may also show small nodules which project slightly, are of the size of a small pea and of a reddish color. In most cases of generalized blastomycosis the remaining viscera present the same nodular masses as do the lungs, but they are apt to be of smaller size. It may be also, as in Cleary's case, that the organs show amyloid degeneration, a condition very likely to occur as a result of the chronic suppuration in these cases.

Symptoms.—The clinical picture presented in cases of generalized blastomycosis varies in accordance with the distribution and predominance of the lesions; but in all thus far reported there were symptoms pointing to lung-involvement. As usual these consisted of cough, expectoration, anorexia, emaciation, loss of strength, fever and acceleration of the respiration. In some there were clearly recognizable symptoms of a general infection, but in Cleary's case this was absent as judged by the temperature, the main features having been those of a chronic pulmonary and chronic renal affection.

Cough.—This is present whenever the lungs are the seat of the blastomycosis, but its frequency and severity are variable. As a rule it is not very troublesome, yet may be distressing, as in 2 of the recorded instances. Since in 2 other of the 5 reported cases this symptom was not mentioned, although the autopsy demonstrated marked lung-involvement, it may be assumed that cough was trifling and hence failed to attract notice.

Expectoration.—This symptom like the one just mentioned may be variable but is probably not wholly absent. It is of a muco-purulent or wholly purulent character and may be streaked or tinged with blood, as in 2 of the 3 cases in which sputum was recorded. The expectoration is due to the associated bronchitis and the hæmoptysis was probably owing to the violence of the cough, since the lesions in the lungs do not tend to cavity-formation. The characteristic fungi may be discovered in the sputum as in Eisendrath and Ormsby's case (Fig. 85).

Emaciation.—This is not necessarily pronounced, and yet in Busse and Buschke's case was extreme. In the patient seen by me in Cook County Hospital and described in Eisendrath and Ormsby's report this feature was not at all marked. Indeed, the man seemed to me in a state of good nutrition, considering all the circumstances. The degree of wasting probably bears an intimate relation to the extent and severity of the changes in general and not to those in the lungs.

Fever.—This may be said to be a fairly constant symptom, notwithstanding its absence in Cleary's case. It depends undoubtedly on the extent of pus-formation and therefore on absorption of toxins. In Busse and Buschke's case it was of a distinctly pyemic character. In Cleary's patient, skin lesions and subcutaneous abscesses were not present and even in the viscera abscess-formation was absent. The lesions consisted chiefly of nodular growths and the man was thought to have died of his chronic nephritis and amyloid degeneration; so that conditions favoring absorption of toxins were not present and the absence of pyrexia is readily intelligible.

Chills and Sweatings.—These, like the degree of body-temperature, are determined by the amount of pus, i. e., abscess-formation and hence absorption of toxins. Although definite statements have not been made on this point, it is probable when we take into consideration the character of the pulmonary lesions that these symptoms depend upon the changes elsewhere rather than in the lungs.

On the whole, it may be safely stated, I think, that the symptoms referable to the pulmonary system are of a less severe nature than in pulmonary tuberculosis, particularly miliary pulmonary tuberculosis. They may be the ones especially to attract the attention of the observer or they may be subordinate to those attributable to other visceral and skin lesions.

Dyspnœa.—This may or may not be experienced, depending on the extent of the pulmonary changes. In most instances the respirations are accelerated, as was noted in some of the recorded cases.

The course of the disease is essentially chronic, tending to persist for a term of years. Thus, in Eisendrath's case the disease began in February, 1904, and admission to the hospital was not sought until a year subsequently. In this case it is noteworthy that cutaneous lesions did not appear until four months after the onset of his obscure pulmonary symptoms. There may be periods of improvement depending upon treatment, but in all cases thus far reported the tendency of the complaint is to progress more or less steadily downward.

Duration is variable. Busse and Buschke's patient had cutaneous lesions for a period of seven years, but the man died in about eighteen months after the development of symptoms pointing to general or systemic infection. In Cleary's case the history was of a duration of months, not years.

Physical Signs.—There appears to be nothing in the physical findings on the part of the lungs that especially marks blastomycosis of these organs from other pulmonary disorders. All reports to date agree in the statement that examination of the lungs showed dullness and bronchial breathing on

one or both sides; and in Walker and Montgomery's case as we
Cleary's the findings were thought to indicate tuberculosis.

Inspection.—In the majority of the instances thus far record
have been skin lesions which, to the experienced dermatologist, be
true nature of the disease. These consist of elevated areas having a
zone or ring and from the center of which a small amount of pus
squeezed. There are also abscesses that penetrate more deeply into
or beneath a ligament, while in some instances lesions are covered o
brownish-red crust or scab, or there may be cicatrices where the lesi
healed. Subcutaneous nodules are also present.

In the facies of the individual there is nothing to point to hi
a pulmonary affection, unless it be emaciation, and even this may n
all marked. In Eisendrath's patient I was particularly struck by the
of pronounced depression of the apical regions, as would be seen in
vidual with advanced tuberculosis. The expansion of the summit
chest was defective, but aside from this there was nothing especial
worthy.

Palpation.—As might be expected in any case of chronic lung di
this type, there is more or less increase of pectoral fremitus. In the
just mentioned this was apparent at the right apex, whereas over
fremitus seemed to me decreased not alone by comparison but actuall
might well be the result of a thick studding of the pulmonary tis
miliary nodules and consequent compression and inflammatory blo
the alveoli and terminal bronchioles.

Percussion.—The resonance of the lungs is more or less impai
degree of dullness depending upon the number and distribution
nodules. This alteration of the percussion-note is more noticeabl
apices and is quite likely to be more pronounced and extensive in o
In this respect the disease both agrees and disagrees with pulmonar
culosis. It is not uncommon to see tuberculous patients with bilate
ness, etc., but this is not usual in those who present no more indic
advanced phthisis than did the man I examined. Yet, in him, bot
lobes showed distinct though not intense dullness.

Auscultation.—The breath-sounds are correspondingly altered.
exhibit more or less of a bronchial quality, but in the patient alrea
referred to, they were enfeebled rather than intensified, especiall
left apex. Râles were not detected and, judging from the *post*
findings recorded, râles would not be produced unless by the bronchiti
is an associated feature in the morbid anatomical findings. There
would not be the numerous adventitious sounds so frequent in
pulmonary tuberculosis.

Diagnosis.—This must depend upon the discovery of blastom
the sputa. In the absence of these the diagnosis of this pulmonary
can be only inferential. Still it must be acknowledged that the re
of characteristic skin lesions in an individual suffering with cough,
or muco-purulent expectoration in which the fungi are not foun
tolerably good evidence in favor of blastomycosis of the lungs.

CHAPTER XXXII

PULMONARY SYPHILIS

Introductory.—This subject no longer possesses the importance once ascribed to it. This must be attributed to the fact that since the discovery of the tubercle bacillus and the establishment of the unity of tuberculous consumption it has become probable that many of the cases once recorded as syphilis of the lungs were in reality tuberculosis. Furthermore, although, as remarked by Samuel West, there is no reason why syphilis should spare the lungs any more than the other viscera, and does not spare them, still cases of indubitable lung changes of this nature are absolutely and relatively very rare.

Indeed instances of gumma of the lungs (the only manifestation of the disease in the lung capable of conclusive demonstration as specific) are so uncommon as to attract special attention and interest from pathologists and by them are exhibited as curiosities. Still more rare are cases susceptible of clinical diagnosis or such as manifest a disappearance of symptoms under antisyphilitic medication and hence may be assumed to be specific.

Thus, West is authority for the statement that out of 6,000 syphilitic subjects admitted to the Hospital of Copenhagen during a period of seven years but 2 furnished clinical evidence of pulmonary syphilis. Moreover, of 18 autopsies of adults dead of acquired syphilis, specific disease of the lungs was found in 3 instances, while out of 105 *post mortems* on children with congenital syphilis the lungs showed the disease in but 4 instances. Lenhartz states that his teacher, the great pathologist E. Wagner, could recollect but a single instance in which he was able to make a clinical diagnosis of pulmonary syphilis and in which a cure followed energetic antiluetic treatment.

Kingston Fowler states that he was able to discover but 12 specimens of undoubted pulmonary syphilis in the museums of the London hospitals and of the Royal College of Surgeons. The French have paid much attention to this form of lung disease and appear able to recognize the affection clinically, while Dieulafoy distinguishes five classes of cases according to their symptomatology. German writers have also devoted great attention to the subject and have reported cases.

When, however, one reads the contributions to this subject he is pretty sure to get the impression that although clinically recognizable disease of the lungs may be discovered in individuals who are subjects of lues, still it is not certain that the pulmonary changes are not tuberculous and secondary to syphilitic infection. Such is particularly the case with instances of chronic ulcerative phthisis. As a matter of fact such is the conservative view held by many of the authors who record such cases.

Not only is there much uncertainty and obscurity concerning the disease in its clinical aspects, but even pathological anatomy has not yet been able to settle all the questions that interest the clinician. That gummata may develop in the lungs we know conclusively, but whether the interstitial changes attributed to syphilis are not in most instances of tuberculous origin, or whether, as claimed by some, these fibroid changes affect the middle and base of the lungs by preference or exclusively are questions that as yet have not been definitely answered.

Ætiology.—As is the case in other organs, syphilis of the lungs may be either congenital or acquired. In the former event the child is usually still-born or survives for but a very short time. As will be seen in the morbid anatomy the lungs are likely to be so extensively diseased as to render respiration impossible. There are a few cases on record, however, of individuals with inherited syphilis of the lungs who have lived to reach adult age.

When this disease of the lungs develops in persons who have acquired lues it is in the tertiary stage of the disease. Consequently the pulmonary changes attributable to syphilis generally occur a number of years after the initial sore. We speak of them as a late manifestation, but it is well to remember that by late is not necessarily meant late in point of time.

Tertiary manifestations or, as we say, laté manifestations of the disease ordinarily appear after a lapse of three, five or more years, and yet instances are now and then seen which run an exceptionally rapid course, from the early to the late symptoms in months or a year or two.

Accordingly the changes produced in the lungs by lues belong to the tertiary manifestations. Ruhemann reported a case which developed dullness, bronchial breathing and moist râles at the left apex down to the fifth rib six months after the initial sore and which was improved by specific treatment. Fraenkel doubts the syphilitic nature of this case, however, and states that pulmonary syphilis develops in from two to five or more years as in one of Zinn's seven cases which occurred eight years after infection.

An exception to the above rule is found in the bronchitis occasionally observed in syphilitic patients. Such a bronchial catarrh may be seen in the early period of the secondary stage and hence Schnitzler lays stress on the importance of recognizing the possibility of a syphilitic catarrh of the bronchial mucosa just as much as the more serious forms of the late stage. Fraenkel regards such a catarrh as nothing more than a continuation of the catarrh from the mucosa of the upper air-passages. He admits, however, that there is much in the view that it may be the equivalent of the cutaneous erythema or roseola seen in the commencement of the secondary stage.

Morbid Anatomy.—In cases of *congenital* syphilis the disease may manifest itself in the lungs in either of two ways, namely, as gummata or as the so-called white pneumonia (pneumonia alba). The gummata differ in nowise from those found in the acquired cases and need no description here.

Pneumonia alba causes the affected lung or a portion thereof to appear as a dense grayish-white mass which is wholly devoid of air when the fœtus is still-born, while in case the child may have survived for a brief period the lung may be inflated in such parts as are not solidified. The entire organ may display this change or the pneumonia may be scattered in isolated patches throughout the lung.

On microscopic examination the alveoli are found filled with desquamated epithelial cells that have undergone degeneration and their walls are thickened.

In *acquired* cases of syphilitic disease of the lungs two varieties are distinguished as was pointed out by Virchow. In one there are gummata which vary greatly in size and number. They may differ in size from that of a hazelnut to that of a walnut or of a small Tangerine orange. There may be one or several such masses scattered throughout the organ. In their early stage they are elastic and of a whitish or reddish-white color. Later on they undergo caseation and soften or become of firmer consistence. Accordingly, gummata appear so like tuberculous masses as to make their differentiation often a very difficult matter.

These syphilomata are surrounded by a dense fibrous capsule and have their seat in the interstitial connective tissue. By some it is said that gummata may soften and breaking into a bronchus be discharged leaving an ulcerated cavity behind. Others as Osler doubt such an occurrence and believe that when such a breaking down takes place it is the result of the action of tubercle bacilli. A more common fate of gumma is to become converted into a dry firm cheesy mass or to undergo transformation into interstitial tissue.

The second or interstitial form of pulmonary syphilis is believed to be more frequent, and indeed is often met with *post mortem* in old syphilitic subjects. The fibrous tissue is seen most commonly and abundantly at the hilus of the lung whence it may extend as dense bands into the interior along the air-tubes and blood-vessels. It may be quite general and lead to extensive pulmonary fibrosis and shrinkage, or it may occur in scattered areas. There are very commonly, also, dense firm adhesions in the pleural cavity. The disease is usually unilateral.

This interstitial hyperplasia displays no characters that serve to distinguish it from fibrosis due to tuberculosis unless it be its tendency to be confined to but one lung and to occupy the middle parts of the organ. Both the interstitial and the gummatous forms may occur together.

Symptoms.—The clinical picture produced by syphilitic lesions within the lungs is somewhat indefinite and depends largely upon the extent of the changes. It is doubtful if a single gumma would be likely to occasion symptoms unless perchance it should be of such size or location as to exert pressure on important blood-vessels or large air-tubes. For the most part gummata within the lungs are an accidental discovery of the *post-mortem* room. Likewise, the existence of interstitial thickening at the root or in the middle of the lung is hardly likely to produce symptoms unless so extensive as seriously to compromise the respiratory capacity. There may be dullness and bronchial breathing in the interscapular region and these signs may enable one to recognize the changes that are responsible for them, but subjective phenomena may be trifling.

There is a class of cases, however, in which the clinical picture of chronic phthisis is observed and which are described in text-books as instances of pulmonary syphilis. They occur in individuals who present evidences of tertiary lues or have been infected a number of years before, but whether the symptoms are in reality due to the specific disease is still an unsettled question. This class of cases will be taken up later on.

In typical syphilis of the lungs in the adult there are but two symptoms that can properly be attributed to the lesions *per se*. These are *cough* and *dyspnœa*. In the bronchitis that occurs in the early secondary stage, cough and expectoration are present as in any other case of bronchial catarrh. There is nothing peculiar in it, however, unless it be that it yields to mercury and iodides.

In the tertiary manifestations *cough* varies in intensity according to the extent and character of the changes. If gumma be present this symptom may be insignificant, but no rule can be laid down for all cases, as in some an associated chronic bronchitis or a laryngeal or tracheal stenosis with accumulation of secretions below may be responsible for a very troublesome cough and dyspnœa. If *expectoration* is present, it is catarrhal or mucopurulent.

When interstitial changes in the lungs are extensive they are apt to lead to retraction of the lung and bronchiectasis with or without narrowing of a bronchus. In such a case the condition, as far as symptoms are concerned, is one of fibrosis and the clinical picture is such as belongs to that affection.

Dyspnœa is the symptom usually said to be occasioned by this interstitial form of pulmonary syphilis, and what is especially worthy of note is that it may be out of all proportion to any discoverable physical signs. Expansion of the affected lung may be lessened, the side may look more or less retracted, there may be either distinct or indefinite impairment of percussion-resonance and the breath-sounds may be harsh and more or less of a bronchial quality, or they may be diminished, and by some writers it is said there may be dry parchment-like or creaking sounds. There is nothing in all these, however, to render them peculiar to syphilis rather than to a chronic inflammatory process of a very different nature.

In contrast to the foregoing cases in which the lesions are gummatous or fibroid and symptoms are correspondingly characterless, others are encountered that present a marked phthisical history and symptomatology. These are designated, therefore, syphilitic phthisis. Many of them are tuberculous as proven by the discovery of tubercle bacilli in the sputa and of tuberculous lesions *post mortem*. For this reason one may not unreasonably question if all instances of chronic ulcerative phthisis seen in syphilitic individuals are not in reality tuberculous.

A case in point is the following, seen as it happened since the writing of this chapter was begun. A physician of fifty-three consulted me in June, 1905, because of cough and failing health. He had been healthy up to three years before, when he contracted syphilis from a patient. Mercury and iodides were taken for only a few months and not very energetically. Two years ago his urine showed a trace of sugar and he dieted accordingly. About a year ago he began to cough and since that time he has lost considerable weight.

Physical examination showed a tall, gaunt man whose movements were somewhat tremulous and who displayed the alar type of chest in a well-marked degree. There was noticeable atrophy of the musculature in general and in particular of the apical muscles of the chest. The peripheral arteries were moderately stiff and the pulse was feeble, 116 to the minute.

The right half of the thorax was dull from apex to inferior angle of the scapula and in front down to the third interspace. Below the middle of the clavicle was a zone of high-pitched tympany, and Wintrich's change of pitch could be elicited though not very distinctly. The left apex was also less resonant than it should have been, but the rigidity of the thoracic walls and the want of normal resonance on the opposite side made rather uncertain the results of percussion.

Over the area of dullness on the right the breath-sounds were bronchial, particularly over the cavity where the voice was bronchophonic but without that "after-breath" I have mentioned in the diagnosis of pulmonary cavities. In other places, especially behind, the breathing was feeble and semibronchial rather than sharply bronchial and voice-sounds were feebly bronchophonic. Nowhere were moist râles to be elicited by forced inspiration or cough.

The temperature at 2 P.M. the hour of the examination, was 98.2° F. in the mouth. He did not complain of night-sweats. He was required to cough and expectorate with the result that a small amount of fluid purulent sputum was obtained which on careful microscopic examination did not show bacilli.

The urine voided in the office showed no albumin and but a trace of sugar.

Now here was a case which suggested the possibility that the symptoms were due to syphilis of the lungs. Still, doubt of its syphilitic nature was entertained, chiefly because of the apical location of the changes and the suspicion of beginning involvement of the opposite lung. Accordingly, he was instructed to send in a morning specimen of sputum for further examination. This was done and careful search at length revealed half a dozen unmistakable tubercle bacilli.

When one studies the cases reported as instances of pulmonary syphilis which, to all intents and purposes, are cases of chronic consumption he is struck by their chronicity and the association of phthisical symptoms with evidences of their luetic origin, such as gummata or old scars in other organs, or stenosis of larynx, trachea or bronchi. The signs of disease on the part of the lungs do not, as a rule, differ from such as are discovered in cases of fibroid tuberculosis or cirrhosis with bronchiectasis. This is brought out plainly in the cases presented by Fowler, one of which will be reproduced in these pages.

The recognition of their real nature during life must depend largely upon the persistent absence of bacilli, while at the autopsy there must be a want, on minute search, of all evidence of tubercles. The cases narrated in Fowler and Godlee's work are thought to fill these requirements and hence may be regarded as good illustrations of syphilitic phthisis.

"Case 1—Charles N., aged thirty-eight, bricklayer. In 1892 he suffered from cough, with expectoration and pain on the left side of the chest. In 1893 he had night-sweats and dyspnœa. He has not lost weight, and, beyond an occasional streak of blood in the sputum, there has been no hæmoptysis. From January to May, 1894, he was an in-patient of the Brompton Hospital, under the care of Dr. Mitchell Bruce; the diagnosis then recorded was, Syphilis (?), tracheal stenosis, chronic bronchial catarrh, induration of the left upper lobe and of the left base with pleural adhesions over that area, cicatrization of the soft palate, and adhesion of the right posterior pillar of the fauces to the back of the pharynx. There were no bacilli in the sputum. He continued fairly well until October, 1894, when he expectorated a large quantity of offensive purulent material for two consecutive days. Cough was very severe at this period. His health subsequently improved, and remained so until February 20, 1895, when, in the course of a few days, he brought up about a quart of blood-stained sputum. Hæmorrhage then ceased and dyspnœa diminished. On March 5th cough and dyspnœa increased and he became seriously ill, with constant headache and slight delirium. Œdema of the feet subsequently supervened. On March 15, 1895, he was admitted to the Brompton Hospital under the care of Dr. Percy Kidd. On admission he was reported to be fairly well nourished; stridor marked; cough severe. Right lung resonant everywhere; breath-sounds much exaggerated, expiration prolonged. Loud hoarse inspiratory and expiratory stridor all over the lungs; sibilant rhonchi general. Left lung.—Expansion much diminished; resonance much impaired, front and back; breath-sounds weak; expiration prolonged; fine crackling râles over the whole of lung; vocal fremitus and resonance diminished. Expectoration profuse and difficult to expel. No tubercle bacilli found. Temperature 99° F. It varied between that point and 96° F. during the time patient was in the hospital. The dyspnœa gradually increased and death occurred on April 10th.

"*Post mortem.*—Scars on tongue, glans penis, and scrotum; and adhesion of skin to left testis. Marked thickening of left tibia. Larynx normal. Trachea narrowed at the lower end. Recent ulceration from cricoid downward for two inches; below this, down to point of bifurcation, there was extensive scarring of the cartilaginous portion; and also at its line of junction with the posterior wall. The submucous tissue was extremely thickened. Cartilages bare in several places. The main bronchi were much scarred and showed extreme narrowing. The bronchus to the left upper lobe was impermeable to a probe. Right lung.—Old pleural adhesions over the upper lobe, recent pleurisy with effusion at the base. Emphysema, with a reticular fibrosis, especially around bronchioles. Deep in the upper lobe at the edge of one of the main bronchi there was a large fibroid mass, with fibroid radiation into the surrounding tissue; in other parts two small, hard, raised masses, one with fibrous strands running up to it. Base solid from broncho-pneumonia. No appearance of tubercle. Left lung.—Upper lobe extremely contracted, containing no normal tissue. It consisted of deeply pigmented blackish-gray fibrous tissue surrounding the openings of bronchial tubes, and bronchiectasis. At the center there was a smooth-walled cavity about the size of a small chestnut into which a bronchus opened. No appearance of tubercle. Lower lobe emphysematous, with reticular fibrosis along the margin and at the base. Bronchi dilated, but not to a marked degree. About the center point of the outer margin there was a small nodule, probably a gumma, white and firm, and surrounded by a pigmented fibrous capsule. The extreme base consisted of indurated fibrous tissue extending from the pleura to a cavity, the size of a marble, into which a small bronchus opened. From this cavity fine fibrous bands radiated in all directions, producing extensive fibrosis of the surrounding lung. Perihepatic and splenic adhesions. Liver scarred and nutmeg. Spleen contained several calcareous masses surrounded by a fibrous capsule. Testes fibrous."

From the foregoing recital it is seen how similar are the symptoms to those of an ordinary case of chronic fibroid tuberculosis, with the exception that there was not so much wasting, while fever and sweats were not so conspicuous a feature as in advanced tuberculosis. Dyspnœa became severe toward the end of the illness and yet, on physical examination, there were not the signs of extensive excavation as is so apt to be the case in ordinary consumption.

Cough and expectoration partake of the characters seen in bronchiectasis and, when paroxysmal, are usually referable to stenosis of the lower air-passages. Because of the retention and decomposition of the secretions the sputa and even the breath may give forth a fœtid odor. *Hæmoptysis* is not one of the common symptoms, being by some declared rare. *Pain* is also not common and yet may be complained of and then may be pleuritic, as shown by the discovery of a recent pleurisy in the case just narrated.

On the whole, one should remember that the symptoms and physical signs are those of fibrosis and consequent diminution of respiratory capacity rather than of lung destruction as in most cases of pulmonary tuberculosis. Herein, in fact, seems to lie the chief distinction between the two classes of cases.

Physical Signs. Inspection.—The appearance of the chest is likely to be altered in accordance with the extent and situation of the fibrosis but will scarcely present the retraction of the apices seen in tuberculosis. If the lungs are extensively diseased their expansion will be diminished and there may be unilateral retraction like that seen in cases of cirrhosis from other cause. It is chiefly in cases of so-called syphilitic phthisis, however, that inspection affords evidence of pulmonary disease.

Palpation.—The results of palpation also depend upon the kind and extent of the changes. Vocal fremitus will probably be increased in certain areas over which dullness exists or where the lung may have undergone ulceration. In some cases of pronounced fibrosis with pleuritic thickening one may be surprised to find that pectoral fremitus is actually decreased. Such increase of vibration as may exist is likely to be found over the back about the root and central portions of the lung.

Percussion.—Pulmonary resonance is likely to suffer more or les
tion by becoming dull where fibrosis exists, or tympanitic over bronc
or other cavities or where emphysema is present. According to some
ities dullness occurs chiefly in the interscapular regions about the
at the base, although, as shown by Fowler's case, it may be present
upper lobe. The presence of pleural adhesions is also shown by imp
of pulmonary resonance at the bases. Gummata are generally too sn
scattered to occasion dullness.

Auscultation.—The breath-sounds are likely to be harsh and in
or distinctly bronchial, according to the nature of the pathological
They may, however, be diminished in areas peripheral to stenosis of
tube. In some of the reported cases there has been inspiratory and
tory stridor due to narrowing of larynx, trachea or main bronchi.

The voice is apt to be bronchophonic. The absence of moist râl
ative of softening has generally been commented on by writers.
there may be rhonchi, or the ear may detect indefinite creaking sound
ated by adhesions. It is particularly significant if the abnormal sou
detected in the middle and lower parts rather than in the apices.

On the whole, it may be stated that there is nothing in the
signs especially characteristic of pulmonary syphilis. They are all
may be found in other diseases that lead to interstitial thickening
without bronchial dilatation and the changes of chronic bronchial
It is especially worthy of note that evidences of extensive lung des
are not discoverable. In this respect, therefore, these cases differ from
ulcerative tuberculosis.

Diagnosis.—This is possible only in a limited number of cases a
then conditions must be favorable and the diagnosis must be arrive
exclusion. The first of these conditions is a clear history of previous
which has been inadequately treated or there must be indubitable
lues in its tertiary manifestations.

Given such an anamnesis or such evidence of late syphilis in an in
with pulmonary symptoms, one may suspect the presence of lung
but cannot make an absolute diagnosis, for we know tuberculosis
uncommon in old syphilitic subjects. The sputa must be repeate
carefully examined for bacilli should the germs not be discovered
Yet, even the failure to recognize tubercle bacilli does not of necessit
a case as nontuberculous.

In a case of chronic tuberculosis that has gone on to formation o
or fibrosis the expectoration may be purulent and fail to contain ba
these latter may be present very sparingly and only occasionally. N
less, the failure to discover tubercle germs after repeated and pai
search over a considerable period of time greatly strengthens the i
to be drawn from the history and signs of late syphilis.

If, in addition, the examination of the chest discloses signs of dise
seem rather anomalous as regards their possible tuberculous origin.
point clearly to fibrosis of other parts than the apex, the suspicion
still stronger. This suspicion becomes a fairly reasonable certainty if

of the larynx, trachea or bronchi is also present. There are clinicians who would probably make a diagnosis of pulmonary syphilis from such a conjunction of data, but I must confess frankly that even under all the circumstances above given I should prefer to make a tentative rather than a positive diagnosis.

As a final resort, one might institute antisyphilitic treatment with a view to establishing a therapeutic diagnosis in such doubtful cases. I cannot dispute those who maintain that a marked improvement in general and local symptoms following the energetic use of mercury and iodides makes fair the assumption that the case is syphilitic. As emphasized by West, we know that benefit has been said to accrue even to tuberculous patients from the employment of the remedies just mentioned, and hence, although the therapeutic test should be tried in a suspected case it cannot be declared an absolute proof of its nature.

The subject is beset with many difficulties since it involves very nice differential diagnosis on some points considered of valuable aid in the determination of the matter in hand. For example! It has been stated that the discovery of tracheal or bronchial narrowing strengthens materially the inference drawn from other data. Still, in estimating the value of bronchial stenosis one must carefully determine, if possible, whether or not it be caused by pressure from an aneurysm. Now, aneurysm is common in old syphilitic subjects and, in fact, may be responsible for a clinical picture closely resembling the so-called syphilitic phthisis. In any suspected case, therefore, it would be well to subject the individual to an X-ray examination.

From the foregoing it is obvious that rare as are cases of pulmonary syphilis from a pathological standpoint, they must be still less frequent in the experience of the careful clinician. As remarked by Dieulafoy, one should always be on the watch for such cases and many times he may suspect he has to do with one of them, but in the diagnosis he should always exercise the greatest reserve. I have seen a number of cases in which syphilis of the lungs seemed probable and yet, either because of the discovery of tubercle bacilli or for some other reason, I have never yet been able to make such a diagnosis.

Prognosis.—This cannot be considered very favorable, even if energetic treatment with mercury and iodides be instituted. The general health may be benefited but the changes suffered by the lungs cannot be removed. Gummata may be dissolved, but the fibrosis, stenoses, bronchiectases, etc., will remain.

Recurring hæmoptysis in a syphilitic patient has been known to cease after the prolonged use of large doses of iodide of potash, but in many
cases symptoms have only been alleviated, not removed. Neve
patient should always be given the benefit of the doubt in the
a relative cure may be established. The fact that individuals suff
so-called syphilitic phthisis have come to autopsy when under
clinicians of great skill and experience proves sufficiently the ser
of the disease.

Infants who are the subjects of inherited syphilis of the lung ally still-born. On the other hand, instances are on record of suc having reached adult age. In such cases, therefore, the prognosi upon the extent and nature of the pathological changes. If the chi long enough to demonstrate its ability to breathe, hope may be e of its living if appropriate treatment be begun.

Treatment.—It goes without saying that this should be th use of mercury and iodides. Fortunately the modern management c is such as to render the liability to pulmonary lues less than former this fact, as suggested by Fraenkel, may be attributed the increas of such cases.

In addition, the employment of modern hygienic measures adopted. These include fresh air, nourishing diet and strict medi vision, the same as in cases of chronic pulmonary tuberculosis. detailed description of these the reader is referred to that chapter.

CHAPTER XXXIII

ECHINOCOCCUS CYST OF THE LUNGS

Ætiology.—Echinococcus or hydatid disease, unlike the affections that have been considered, is due to the action of an animal parasite, the tenia echinococcus. This variety of tapeworm infests the dog and wolf and hence echinococcus disease in man is most common among communities in which the dog is a frequent household pet. It is more rarely met with in the United States and Great Britain than in some other countries, especially Australia, Iceland and Germany. The disease occurs far less frequently in the lungs than in other organs, particularly the liver, and hence this form of pulmonary disease is one of great rarity in this country.

Of 85 cases of hydatid disease collected by Osler the lungs or pleura were affected in but 6, or about 7 per cent, which agrees fairly well with the percentage of pulmonary cases found by most other writers. Neisser found the lungs to be the seat of the disease in 7.44 per cent, while the combined figures of Davaine, Cobbold, Finsen and Neisser give 8.3 as the percentage of pulmonary cases (West). On the other hand, the figures collected by Thomas in Australasia show the lungs involved in 16.56 per cent (Fowler). This is an exceptionally high percentage, since of 1,897 cases of echinococcus disease collected by Thomas from all parts of the world the lungs were affected in 11.59 per cent. The liver is far more often affected, its relation to hydatid disease of the lungs being as 6.5 to 1 in Europe and 4 to 1 in Australasia (Fowler and Godlee).

The invasion of man by this parasite is accomplished in consequence of the discharge in the excrement of the dog of many hundreds of the ova of the tenia echinococcus and of their lodgment upon edible plants or in water. Consequently, it is through the ingestion of infected water or uncooked plants that the ova gain access to the alimentary tract of human beings and the relatively frequent localization in the liver. It is also very possible that the eggs of the worm may invade the system of an individual through the inhalation of dust. It is this circumstance that may explain the relatively greater frequency of hydatid disease of the lungs in Australasia, since the dryness of the atmosphere as compared with Europe would favor the occurrence of dust.

It is generally stated that the ova are taken into the gastro-intestinal tract and that thence the embryo is carried in the blood-stream to the lungs. Fraenkel doubts the likelihood of the larvæ being able to pass the capillaries of the portal vein and thus to reach the inferior vena cava. He thinks it far more reasonable to assume that they penetrate the wall of the pharynx or œsophagus, enter the tributaries of the superior vena cava and by this vessel are carried to the right heart and thence into the pulmonary artery and lungs; or that the embryo passes down the intestines and, at length piercing the wall, enters the branches of the inferior hæmorrhoidal veins which discharge their contents into the inferior vena cava without passing through the liver, and the embryo is so carried to the right ventricle and lungs. Without wishing to deny the possibility of this mode of ingress, I yet maintain that there is in this no satisfactory explanation of the frequent invasion of the human lungs in Australasia.

The very fact that in this part of the globe the inhabitants show a markedly greater percentage of cases of primary echinococcus of the lungs presupposes some other channel of infection. According to Leukart's measurements the ova of this worm have a diameter of 0.017 mm. to 0.03 mm. which, as remarked by Fraenkel, is twice that of a human red blood cell; so that their size precludes the possibility of their being transported by the cellular ingredients of the blood, but is, on the other hand, minute enough to favor their conveyance in the dust of the atmosphere. Nevertheless, the inhalation of the ova is far less likely than their ingestion along with food and drink, and hence the relative frequency of the liver as the seat of the disease.

Secondary involvement of the lungs and pleura is easy of explanation and is favored by hydatid disease of the liver. The cyst penetrates the diaphragm and forces its way into the pleural cavity or through this into the lung. Hence it is that the right lung is a more frequent seat of secondary echinococcus disease than is the left.

Morbid Anatomy.—Echinococcus disease of the lungs appears as cysts or hydatids which may be either single or multiple. The cyst consists of a structureless, stratified envelope of which the outer layer is known as the ectocyst and the inner as the endocyst. This latter is granular and has the property of reproducing other so-called daughter-cysts by a process of budding, and hence it is also designated the parenchymatous layer.

The contents of the hydatid consists of a transparent, nonalbuminous fluid rich in chloride of sodium and containing, as its distinguishing characteristic, the salts of succinic acid. Scolices with their characteristic hooklets (Plate IX) and secondary daughter or brood cysts may also but not constantly be found within the hydatid. The amount of the fluid contents varies within wide limits, from half a pint in a single cyst to even 180 ounces, as in Vigla's case (W. Fox), which the latter author thinks must have been intrapleural.

As is apparent from what was just said regarding the quantity of the fluid echinococcus cysts may differ much in size as well as in number. When but a single hydatid exists it is usually of considerable dimension and may be so large as to occasion serious pressure-effects, even to the compression of the entire lung. Multiple cysts are also found and, according to Lebert, are more common than single ones. They may be multilocular or may be scattered throughout the lungs. In some instances they have been so minute as to require the microscope for their differentiation from tubercles. The right lung is affected more often than is the left, and the lower lobe is the favorite seat of the disease although the upper lobe does not escape. The hydatids may be intrapleural or confined to the interior of the lung and invade the pleural cavity secondarily by growth or rupture.

As the echinococcus cyst increases in size it causes compression of the surrounding parenchyma which may display no other effect than that of pressure or may show a growth of fibrous tissue surrounding the hydatid and forming an envelope or capsule. Such a fibrous encapsulating zone is said to be less common in the lung, however, than in the liver. Occasional further effects, on the parenchyma of the lung, are inflammation (pneumonia) and gangrene. It is also stated that the organ may become fibroid. Pulmonary tuberculosis may exist as an accidental complication, of which an example will be found in a case to be subsequently narrated in this chapter.

Rupture of the cyst may occur with evacuation of its contents through the bronchi and the mouth; or the discharge may be into the pleural cavity with consequent pneumothorax or empyema. After the evacuation of its contents the hydatid may collapse and the disease become ultimately healed; or infection of the cyst-wall or surrounding lung may take place, and either abscess or gangrene be the result. In rare instances the hydatid may be converted into a puttylike mass by the deposit of lime salts, and the disease be thus arrested.

Secondary echinococcus disease of the lung occurs less frequently than does the primary form (Mosler and Peiper). It takes place most commonly in consequence of the perforation of a hydatid of the liver through the diaphragm into the pleural cavity and thence into the lung, or directly into this latter, by reason of adhesions between the diaphragmatic and pulmonary pleuræ (Fig. 87).

In an interesting case of Fraenkel's reported by Aschoff the left lung and pleural cavity communicated by means of a fistula several centimeters in length with a necrotic cavity nearly the size of two fists, which was situated between the spleen and the diaphragm and contained echinococcus cysts. The primary seat of the disease in this case was in the spleen.

Symptoms.—French writers, and following them Fraenkel, distinguish three stages of the disease. This appears to me to simplify the subject, and hence will be the plan adopted in this work.

First Stage.—This is the period during which symptoms are either quite latent or are such as suggest some pulmonary complaint of which the precise nature is not at all clear. For the most part, the individual remains in apparently good health and nutrition. Fever is absent and emaciation does not occur, although exceptions in these respects may be noted. According to Dieulafoy, the one symptom of importance and significance is *hæmoptysis.* This is usually of small amount, may be repeated at intervals over a considerable period of time and, according to Delgrange, is rarely absent altogether. Owing to the absence of definite lung-findings in this stage the hæmoptysis is very likely to suggest the presence of tubercle in the lung.

Pleurisy is another early symptom on which particular stress is laid by Dieulafoy and which, according to Marconnet, occurs in repetitions and may occasion an exudate that may undergo absorption with astonishing rapidity.

Other symptoms in this stage are not usual and must depend for their development upon the extent and number of the cysts. Thus, it is said that dyspnœa and troublesome cough have been noted.

Second Stage.—This marks the phase of the complaint in which physical signs appear in the lungs. These are areas of dullness with diminution of tactile fremitus and alteration in the breath-sounds. For the most part these are enfeebled or abolished, although it is stated that exaggerated and even bronchial breathing may be heard. As might be expected from what was said concerning the lower lobes being the seat of predilection, the signs pointing to echinococcus disease of the lungs are to be especially sought for at the bottom of the chest.

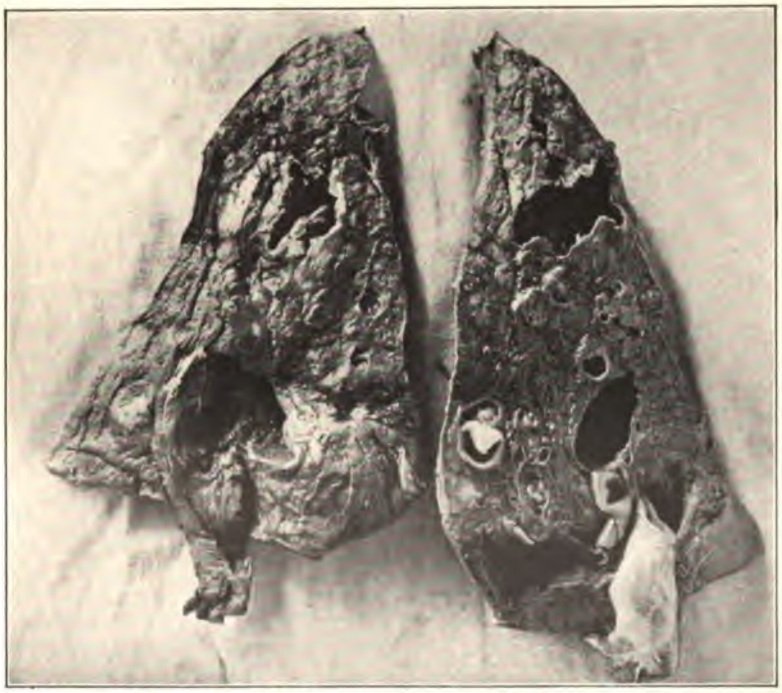


FIG. 87.—Secondary echinococcus cysts of the lung. (Grulee.)

Special weight is attached to the contour of the dull area, this being curvilinear with its convexity upward. Frerichs directed particular attention to this peculiarity in cases of secondary hydatids growing into the lung from the liver, but Fraenkel says it may be present when the tumor is situated in the middle or upper part of the lung. This author also mentions as highly significant the sudden transition from diminished or even absent

stated that the disease may burst into both the pleural sac and the bronchi (Fraenkel).

The bursting of a hydatid of the liver into the lungs and its discharge by the mouth may be evinced by the expectoration of fluid or pus having a yellow hue from admixture with the coloring matter of the bile—bilirubin. This is possible only when communication exists between the cyst and the bile passages. The occurrence of such an expectorate following symptoms pointing to escape of fluid from the lungs should always instigate to a careful search in the sputum for the characteristic hooklets and fragments of cyst-walls, or what is known as the chitinous envelope. In more than one case this has led to the correct diagnosis. It should be remembered that the perforation of the lung by a hydatid of the liver is only likely to take place when the pleural surfaces are adherent. Otherwise the rupture may occur into the pleural cavity.

In this third stage there are symptoms pointing to serious disease of the pulmonary parenchyma, such as chronic cough with more or less expectoration of purulent material, progressive loss of strength and flesh, dyspnœa and fever. This last is not likely to be marked, however, unless the cyst has become infected, a not infrequent occurrence. In such an event the pyrexia may assume a distinctly hectic type.

A very singular phenomenon that has been observed to precede or attend the rupture of the cyst, and on which Dieulafoy and others have laid stress from a diagnostic standpoint, is *urticaria*. The eruption may precede or follow the escape of the cyst contents and has been observed after puncture. From experiments on animals into which the echinococcus fluid was injected with resulting urticaria, inflammation and even death, it has been assumed that the phenomenon is due to absorption of toxins, a toxalbumin (Viron) contained in the hydatid fluid.

Two interesting instances in which this symptom was observed are narrated by Dieulafoy and briefly mentioned by Fraenkel as follows: One was the case of a forty-year-old man who, for four months, had suffered from hæmoptyses and attacks of dyspnœa strongly suggesting asthma. This seemed the more likely as examinations of the chest were entirely negative. Suddenly, during a paroxysm of severe cough, he expectorated a large quantity of purulent sputum which was followed by the outbreak of urticaria. This led to a careful microscopic examination of the sputa with the resulting discovery of characteristic hooklets. The second case was that of one of Dieulafoy's pupils who, for ten months, had suffered from hæmoptysis and loss of weight which occasioned a belief in his having pulmonary tuberculosis. One evening the young man suddenly experienced a chill with urticaria. The next morning he expectorated abundant purulent sputum together with an echinococcus cyst of the size of a nut.

The following case of echinococcus disease, which I had the privilege of examining and which was reported by Dr. C. G. Grulee in *Surgery, Gynæcology and Obstetrics,* October, 1905, is here reproduced because of its unusual interest. It displays well the features of the affection and is therefore also instructive.

"Lizzie P., twenty-seven years old, entered Cook County Hospit
2, 1905, on the service of Dr. Miller. She was a native of Italy, fro
country she had emigrated the previous year. She could talk no
and, as a consequence, the history of the case is very imperfect. We
however, that she had been troubled for four months with cough,
the chest, and an abundant expectoration which, at times, had bee
She complained of nausea and vomiting and had lost forty pounds i
Examination showed a much-emaciated young woman who could n
above a whisper and whose facial expression was that of fear, the e
wide and staring. Tongue dry and fissured. Heart, on percussion
dullness from the right border of the sternum to three-fourths inc
left of the nipple line and above, as high as the third rib. At th
loud, rough systolic murmur was heard, while over the pulmonic a
was also a loud systolic murmur. The lungs showed dullness anteri
posteriorly over the right upper lobe, with some dullness posteriorly
left upper lobe. Low down posteriorly, just to the right of the
column was a small area, about the size of the palm, over which th
sion-note was somewhat tympanitic. On auscultation moist râles a
chial breathing were heard over the right upper lobe, and a few muc
over the middle lobe; some bronchial breathing was heard over the l
and a few moist râles in the left axillary region. In the abdomen
a pregnant uterus reaching to the umbilicus, fœtal movements b
served. Vaginal examination showed the usual signs of pregnancy.

From the lung-findings a diagnosis of pulmonary tuberculosis wa
which examination of the sputum confirmed. During her stay in the
her cough was very violent and she expectorated large quantities of
material. Only once did her temperature rise above 99° F. and at t
was only 99.6° F. Her pulse ranged from 96 to 128. The most
symptom was the extreme dyspnœa. Once her respirations were 2
minute, but this was for a short time only, most of the time the
tions being from 40 to 60 per minute. In order to get sufficient
patient was compelled to sit up in bed and then her breathing w
labored. On the 5th of April the patient coughed up a small cys
one centimeter in diameter. This was unruptured, and showed what
to be subcysts. The fluid of this cyst failed to show any hooklets
large number of small round bodies found within the echinococcus
were seen. The wall of the cyst showed the characteristic laminated
ance. After this the sputum was examined several times for hookl
without success. On the morning of the 10th the blood showed 2,
red blood-corpuscles, 19,000 white blood-corpuscles, of which 88.3 n
were polymorphonuclears, 7.8 per cent large mononuclears, 3.9
mononuclears, no eosinophiles, hæmoglobin 80 per cent (Tall.
patient died suddenly at 6 P.M. the evening of the 10th, and
afterward the child was delivered through a median abdominal i
child breathed once or twice and then died in spite of all efforts t

On the morning of the 12th an autopsy was held. It was n
to open the cranial cavity. Thorax.—No fluid was found in

cavity; the left was free from adhesions, but the right apex was firmly adherent to the thoracic wall. Throughout both lungs could be palpated rounded masses (Fig. 87), some nearly as large as a hen's egg. The left lung crepitated throughout, but the right upper lobe was almost solid and in the middle and lower lobes could be felt hard, shotlike bodies, irregular in outline. On section the left lung showed several large echinococcus cysts throughout both lobes, varying in size from that of a pea to that of a hen's egg. No focus of tuberculosis was found in either lobe. The right lung, on section, showed practically the same distribution of echinococcus cysts, the number being, if anything, slightly greater. The entire right upper lobe had a honeycombed appearance, more marked in the upper than in the lower portion, and gave off a caseo-purulent material. Disseminated tuberculosis existed in the middle and lower lobe. The area of tympany previously described was found to be just below a large cyst located in the upper posterior portion of the right lower lobe. This sign is considered characteristic by Behr. This cyst was removed without rupturing. Examination of the fluid showed the absence of albumin and sugar and the presence of a large amount of sodium chloride. Microscopically, the fluid contained many proligerous vesicles, in which the characteristic echinococcus embryo could be seen. The wall had the characteristic laminated appearance, and on its internal surface were seen numerous daughter-cysts. In removing the cyst the fingers became covered with a thick viscid material, the pericystic fluid. The lung-tissue surrounding the pericystic cavity showed a thin connective-tissue layer, in which there were numerous small blood-vessels. Some of the larger cysts in the right lung had become infected, the cystic fluid being purulent. A smear of this fluid showed a pure culture of streptococcus. Several of the cysts connected directly with the pulmonary vessels, showing that the infection was by way of the blood. In the lung substance numerous typical tubercles were seen. The central portions of the lungs were nearly free from cysts, most of the latter occurring peripherally in the lung substance or just beneath the pleura. The peribronchial lymph glands showed caseous and calcareous degeneration. On examination the heart was found to be free from change, except for the presence of a cyst the size of a pigeon's egg, in the posterior wall of the right auricle. This cyst had ruptured internally, and a clot was found adherent to its endocardial opening. When this clot was removed several daughter-cysts varying in size from a pin-head to a pea escaped into the cavity. The walls of this cyst seemed dryer and harder than those of the lung. A section through the cyst and heart-walls showed that the musculature of the latter had undergone no appreciable change, but there was a thin layer of fibrous tissue between this and the cyst-wall. The stomach and intestines were not removed but careful external examination failed to show any trace of echinococcus. The spleen showed no change. In the kidneys was observed a slight degree of chronic parenchymatous degeneration. The uterus showed the characteristics of a pregnant uterus at six months, the placenta being attached to its posterior wall. No trace of echinococcus disease was seen in other organs than the heart and lungs."

The production of a zone of tympany bordering one of dullness, as in this case, and said to be considered characteristic by Behr can certainly not be peculiar to this disease, for, since it is caused by relaxation of the parenchyma from compression by a mass in its immediate proximity, the tympanitic resonance may occur in any other form of tumor as well as hydatid. After rupture a zone of tympany may be detected over the former site of the cyst, the same as in case of cavity from any other cause.

Auscultation.—Until the disease has reached the stage in which clinical findings become apparent, auscultation yields wholly negative results. When, at length, the affection has attained such a degree as to occasion considerable compression of the surrounding tissue, the breath-sounds are correspondingly altered but do not display distinctive characters. Over the area of dullness there may be diminished or even absent breathing, depending upon the amount of lung substance accessible or pervious to the inspired air. In the portion of the parenchyma surrounding the tumor the conditions favoring the ingress of air are such that the breath-sounds may approach the normal. Hence, as has been already pointed out, it is considered highly significant if the respiratory murmur is found to change suddenly from nearly total or complete inaudibility to loud or even exaggerated respiration.

Similar changes in the voice-sounds are or should be also detected, enfeeblement over the area occupied by the hydatid and intensification where the pulmonary tissue is atelectatic. If râles are present, they are due to associated changes not to the echinococcus disease itself. After rupture and discharge of the cyst, the vocal signs may be such as pertain to a cavity.

Diagnosis.—This can be only inferential as long as rupture and discharge of the cyst contents through the bronchi do not occur, or unless the nature of the case can be established by puncture. If, in either event, a clear fluid is obtained having the characters previously described, the diagnosis may be tolerably assured even without the discovery of fragments of hydatid membrane, daughter-cysts and hooklets. There is no other affection in which fluid of this character may be expelled with the act of coughing. The mere expectoration of abundant purulent material during a paroxysm of cough does not bespeak the bursting of a hydatid; it only suggests it. Careful microscopic inspection of the sputa must then be made, for the possible recognition of the ingredients mentioned.

Likewise, the sudden appearance of yellow expectoration is only suggestive, since it may come from a ruptured liver abscess; however, it warrants a minute search in the sputa for hooklets and cysts or portions of them. The sudden eruption of urticaria either shortly prior to the unexpected expectoration of purulent sputa, or soon thereafter, is so singular a phenomenon and has been observed by such trustworthy authorities as to render this sign of great value. It is not sufficient for a diagnosis, however, in the undoubted absence of the pathognomonic bodies enumerated or of a transparent fluid expectorate which, on chemical analysis, is found nonalbuminous and rich in chloride of sodium and succinic acid or its salts.

On physical signs alone it is very doubtful if a positive recognition of echinococcus disease of the lungs can ever be made. It may be possible, however, when signs of pulmonary disease or hæmoptysis are associated with data pointing to hydatid of the liver or other abdominal organs.

Exploratory puncture of an echinococcus cyst of the lung is said to be fraught with grave danger and is not to be recommended. In corroboration of this assertion Fraenkel cites Maydl's figures as follows: of 11 cases in which this procedure was carried out, all proved fatal within a shorter or longer time thereafter. Five died in from three days to a month and 6 in from a few minutes to twenty-four hours after the puncture. Death is said to occur either with signs of pulmonary œdema or in conjunction with the expulsion of the contents of the cyst through the mouth. These unfortunate sequels are attributed to the circumstance that, in consequence of the extreme tension of the cyst, its contents leak out through the site of puncture. Cough is thereby excited and the expiratory effort incident to this act causes a still greater leakage of the fluid into the pulmonary parenchyma with signs of œdema.

Differential Diagnosis. (1) *Pulmonary Tuberculosis.*—This is probably the disease with which echinococcus is most likely to be confounded. This arises mainly from the similarity of the symptoms in both affections prior to the discharge of the cyst. A positive differentiation in the two first stages can only be accomplished by the discovery of tubercle bacilli, and even when rupture of the hydatid takes place the microscope must settle the nature of the case, since in tuberculosis the discharge, through the mouth, of a lung abscess or acutely caseous focus may occur.

As far as the lung findings are concerned it may be remembered that tuberculosis is an apex disease and echinococcus disease is ordinarily basic. That this difference cannot be relied upon is proven by Grulee's case narrated previously.

(2) *Solid Tumors.*—The main difference lies in the far greater likelihood of pronounced pressure effects in neoplasms. In these, moreover, symptoms pointing to bursting of the tumor do not occur. If sudden expectoration of a large amount of purulent sputa should take place, for any reason, it will not contain hooklets, etc. There may be a great similarity in the clinical manifestations in other respects; but malignant disease is very apt to give rise to pleuritis with an exudate which, when aspirated, is not at all like the contents of a pleural echinococcus.

(3) *Rupture of a Subphrenic Abscess into the Lung and Bronchi.*—The antecedent history is quite different and is likely to be that of a chronic disorder of some one of the abdominal viscera rather than of a chronic pulmonary complaint with hæmoptysis. Careful study of the signs will usually disclose the fact that the cavity is situated between the diaphragm and liver with upward compression of the lung. There are no hooklets or echinococcus cysts in the pus. For other points of difference the reader is referred to works on surgery dealing with subphrenic abscess.

(4) *Primary Echinococcus of the Liver or Spleen.*—Under some circumstances a hydatid of an organ situated below the diaphragm may occasion objective symptoms that render its differentiation from one of the lung very necessary. Particular attention must be given to the shape of the

iver or spleen, as the case may be, to the detection of inequalities utline or even of an elastic, semifluctuating tumefaction and to th ective phenomena. It is also most important to study carefully the f dullness in the lower portion of the chest and the respiratory play liaphragm, since in this way it may be possible to recognize the he tumor's encroaching upon the lung rather than having its prima n it. In most instances, probably, the determination of the questi e answered by rupture and discharge of the cyst through the bronch

(5) *Primary Echinococcus of the Pleura.*—This is almost next possible of differentiation from one of the lung, but fortunately is o infrequency as compared with the latter. Of 25 intrathoracic cysts o nococcus origin, Davaine is said to have seen but one of the pleura appearance of a cyst growing through the chest-wall speaks for ei pleural or extrapleural echinococcus.

THE X-RAY IN DIAGNOSIS.—Very little opportunity has been a most experts in the use of the Roentgen ray for the study of the now under consideration. I find that both Fraenkel and Francis H. W cite the observations of Levy-Dorn and Zadek and hence I quote the fo abstract of their case taken from Williams's work:

"The patient was a robust man; he had been a butcher. In 1897 dyspnœa, hæmoptysis, expectoration of pus sometimes streaked with tubercle bacilli were not found. In November, 1898, echinococci were in the expectoration.

"The signs by percussion and auscultation were not decisive, but an examination showed a shadow about 5 centimeters long and 4 cent broad, with a light interior and a dark edge. This shadow was connected with the diaphragm by a short streak. The diaphragm was pulled up at the junction of this streak or band, and could not contract in full inspiration as well as on the left side. In the left lung there was an oval black shadow about the size of a plum. These shadows were found to have a central position.

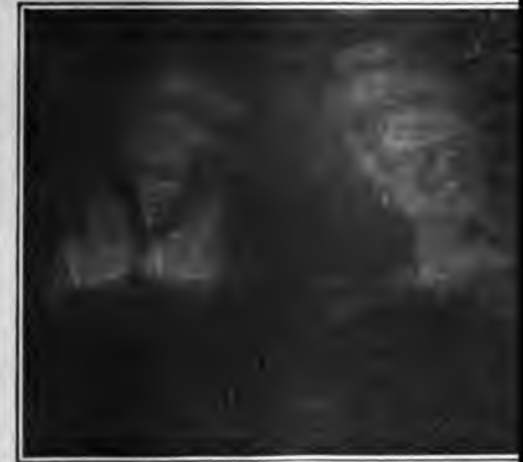


FIG. 88.—Skiagraph of thorax of patient with echi disease. (Fraenkel.)

"The conclusions from the X-ray examination were as follows: the patient had two separate areas of echinococci, one in the right and the left lung. Their central position explained the negative result cussion. As the middle portion of the area in the right lung was light, it was probable that the echinococci were discharged from that side and that the cavity had become filled with air, but were still present in the left lung as indicated by the dark area."

Fig. 88, taken from Fraenkel's work, is a reproduction of Levy-Dorn's original skiagraph and shows well the two areas above described.

Prognosis.—This is most grave as shown by statistics, Davaine having found that death occurred in two-thirds of his collected cases (Fraenkel). Such a termination may be expected in cases not open to definite diagnosis and operative interference or when the disease is not evacuated by way of the bronchi. If the affection can be recognized and subjected to pneumotomy, the results will generally be favorable, Tuffier having reported a cure in 90 per cent of cases operated upon by him. The figures from Hearn are not encouraging. Of 144 cases collected by Hearn, recovery took place in 62 and death in 82; and of the 62 which recovered the fortunate result was attributable to expectoration of the echinococcus in 45 instances. On the whole, the outlook in hydatids of the lungs is not so favorable as in echinococcus of other organs. This becomes very apparent when it is considered that multiple cysts, as in the case I have narrated, may invade both lungs and seriously compromise the respiratory capacity; and even if diagnosed are not open to surgical interference.

Treatment.—This must be surgical or purely symptomatic as the case may be. Pneumotomy is the operation indicated whenever the cyst is accessible. To attempt the evacuation of the hydatid by puncture alone would scarcely be considered nowadays, and is highly objectionable because of the dangers attending the procedure as stated above, under diagnosis.

The symptomatic treatment must consist in measures calculated to fortify resistance and mitigate such suffering or untoward symptoms as may arise. If, after the expectoration of the cyst, a purulent sputum persists it is recommended to treat the condition, i. e., the ensuing cavity, by means of inhalations of various antiseptic remedies the same as in cases of bronchiectasis or fœtid bronchitis, or by incision and drainage when its location is favorable to operation.

CHAPTER XXXIV

MALIGNANT GROWTHS IN THE LUNGS

Carcinoma.—This is by far the most frequent form of malignant tumor the lungs and yet it is absolutely and relatively infrequent. Its frequency relation to carcinoma in general is shown by Reinhardt's figures given by est. Of 545 cases of carcinoma occurring in 8,716 autopsies the lungs

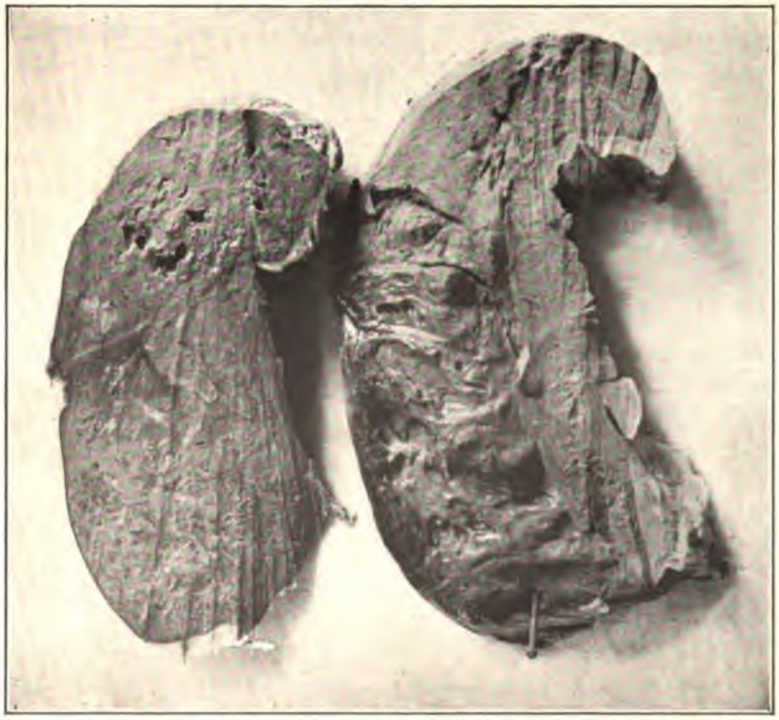


FIG. 89.—Carcinoma invading lung from mediastinum.

ere affected in 74 cases or 13.8 per cent. Of the two forms in which lmonary cancer occurs the primary is very much the less frequent. Thus, 1,000 cases of malignant disease collected by Paessler there were but 16 stances of primary cancer of the lung (Fraenkel), and 4 of sarcoma.

Among 12,307 *post mortems* at Münich between 1854 and 1885 Fuchs found but 8 primary carcinomas of the lung or 0.065 per cent. In contrast to this small number K. Wolf discovered 45 instances of primary lung carcinoma among 20,116 necropsies in the City Hospital of Dresden. Froehlich found 16 instances out of 4,500 autopsies at the Hospital of Urban (Berlin).

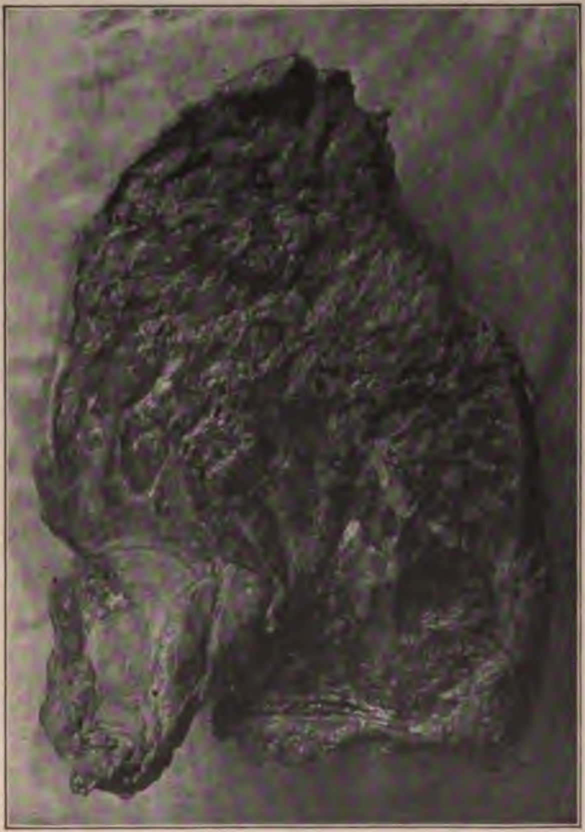


FIG. 90.—Multiple secondary carcinomata of lung.

In contrast to the small percentages of primary carcinoma, the 13.8 per cent of cases of cancer of the lungs discovered by Reinhardt stand out conspicuously and illustrate how much more common is the secondary form. This accords with the statements of all writers, and it is the rarity of a primary growth as well as the fact of its producing more definite symptoms by reason of its larger size which renders this form of greater clinical interest.

Ætiology.—We have no more knowledge concerning the causation of carcinoma of the lungs than of the disease in other parts of the body. Neither do we possess any certain information regarding the factors that predispose thereto. *Age* is believed to constitute one of the chief factors and yet this conception is proven erroneous since figures go to show that it is not confined to any one period of life. Thus, of 61 of Reinhardt's cases in which the age was noted, 32 occurred before the fiftieth year and 29 after as follows: 4 below the age of twenty, 2 between twenty and twenty-five, 4 between twenty-five and thirty, 1 between thirty and thirty-five, 7 between thirty-five and forty, 6 between forty and forty-five, 8 between forty-five and fifty, 6 between fifty and fifty-five, 9 between fifty-five and sixty, 7 between sixty and sixty-five and 7 after sixty-five. Aldhowies reported 1 case in a child of five and a half years (West). Aufrecht also comments on the remarkable fact of its occurrence at a comparatively early age and cites Hasse's 22 cases of which 19 were in persons below forty-nine.

Trauma is held by Aufrecht to be a possible predisposing factor in its causation, and he cites as a noteworthy coincidence the history of "grave trauma" in the 4 cases of primary cancer of the lung seen by him. Georgi and Ebstein are also said to have obtained an anamnesis of injury to the chest-wall in their cases. But how trauma can act as a predisposing factor we cannot explain.

Sex.—Whether this exerts any predisposing influence in itself or not it is noteworthy that primary carcinoma of the lungs has been found in men more often than in women. Thus, of Hasse's 22 cases 17 were in males and but 5 in females. Of Reinhardt's 27 instances 16 were in men and 11 in women. As bearing on the disproportionate frequency of primary lung cancer in the male sex the conjecture may be allowable of a connection between sex and trauma, since the possibility of this latter is greater in men.

Morbid Anatomy.—Carcinoma of the lungs is met with in two forms, the primary and the secondary. Any variety of cancer may exist here as elsewhere but the medullary is the more common. *Primary carcinoma* occurs either as a circumscribed tumor mass or as a diffuse infiltration of the parenchyma. In the former event there is generally but a single growth and yet the lungs may be beset with metastatic nodules as in Ebstein's case in which the primary mass was located in the right lower lobe. This tumor several centimeters in length had converted the wall of a small bronchus into a tube several millimeters in thickness resembling the quill of a raven's feather. The mucosa of the bronchus was implicated in the cancerous growth and had a nodular appearance (Figs. 89, 90, 91, 92).

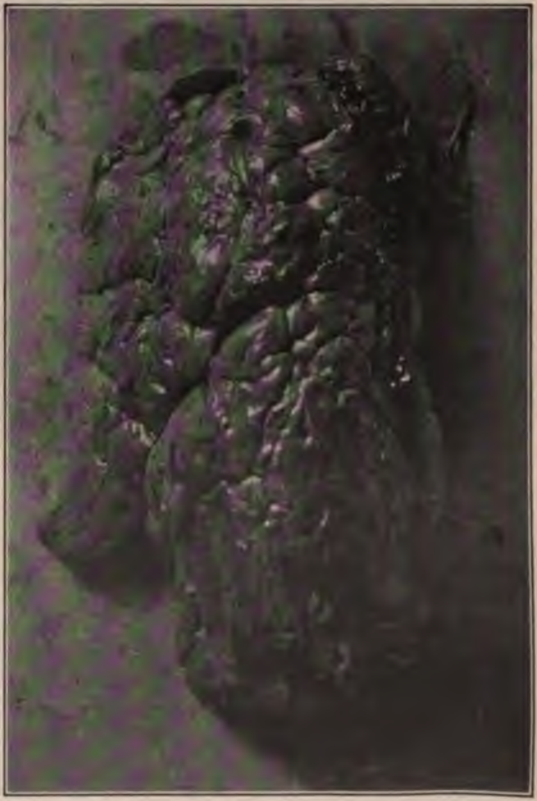


FIG. 91.—External surface of lung shown in Fig. 90.

When the parenchyma is infiltrated with carcinoma the tumor is apt to be of large size and to occupy the whole or the greater portion of a lobe. In the great majority of cases the new growth has its origin in a bronchus (Fraenkel), but is only very exceptionally confined to the bronchus or to its immediate vicinity. From its original point of departure the cancer spreads either by direct extension into neighboring alveoli or along the lymph channels in the outer layer of the bronchial wall. In either event it may give an appearance of an infiltrating cancerous growth (Figs. 89, 90, 91 and 92).

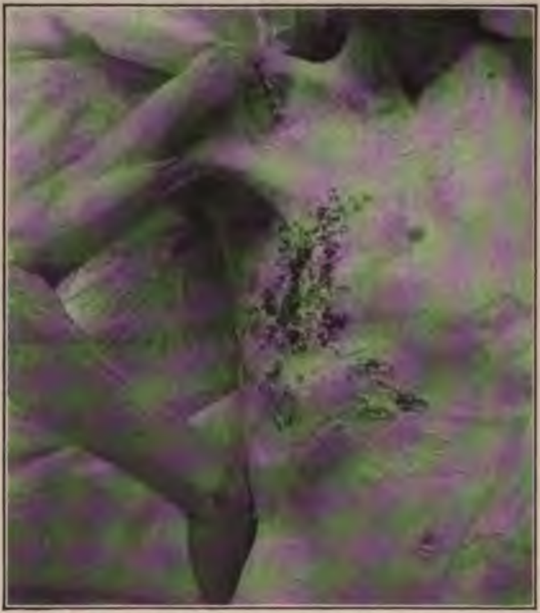


FIG. 92.—Carcinoma of skin primary to that in lung shown in Figs. 90 and 91.

There is much doubt as to the histogenesis of pulmonary carcinoma. Some, as Fraenkel, hold that it originates in a transformation or proliferation of the epithelial cells covering the bronchial mucosa or those lining the mucous glands of the bronchus. From there the tumor may extend either directly or by way of the lymphatics and involve adjacent alveoli thus giving an appearance of having originated in the alveolar epithelium. They do not, however, deny the origin of the cancer in the alveolar cells in a minor proportion of cases.

Aufrecht, on the other hand, holds with Perl, Malassez, Grünwald and others that, in most instances, the cancerous growth originates in an alteration or hyperplasia of the alveolar epithelium. Paessler who has given this subject much attention holds that the question cannot be determined in most cases.

Secondary carcinoma develops in the lungs in several ways, by the blood-vessels as emboli, along the lymphatics or by way of the air-tubes from cancer of tongue, throat, larynx, trachea, etc. When it is of embolic origin the nodules are often of enormous number and of variable size. They may appear as a miliary carcinosis where there are generally found tumor masses in other organs besides the lungs. When the new growth extends to the lungs from the breast, e. g., by way of the lymphatics it may appear as whitish cords of nodular outline ramifying beneath the pleura or in the interior of the organ along the sheaths of the blood-vessels or lymphatics and in close connection with the walls of the lobules.

A carcinomatous growth may undergo softening and thus lead to formation of a cavity which may contain pus. In its breaking down it may give rise to pulmonary hæmorrhage. Finally tuberculosis and cancer may coexist in which event the latter arises in the wall of a phthisical cavity as in cases reported by Friedländer, Wolf and Schwalbe.

Sarcoma.—This is a far less frequent form of malignant disease in the lung than that just considered. All varieties of sarcoma have been met with in this location but the great majority of them are lymphosarcomas. For the most part the new growth is secondary to some tumor in another region of the body. A lymphosarcoma starting in the glands of the mediastinum or at the hilus of the lungs (Fig. 93) spreads along the peribronchial lymph nodes and invades the lung compressing the air-tubes and blood-vessels or occasionally penetrating the lumen of the alveoli themselves.

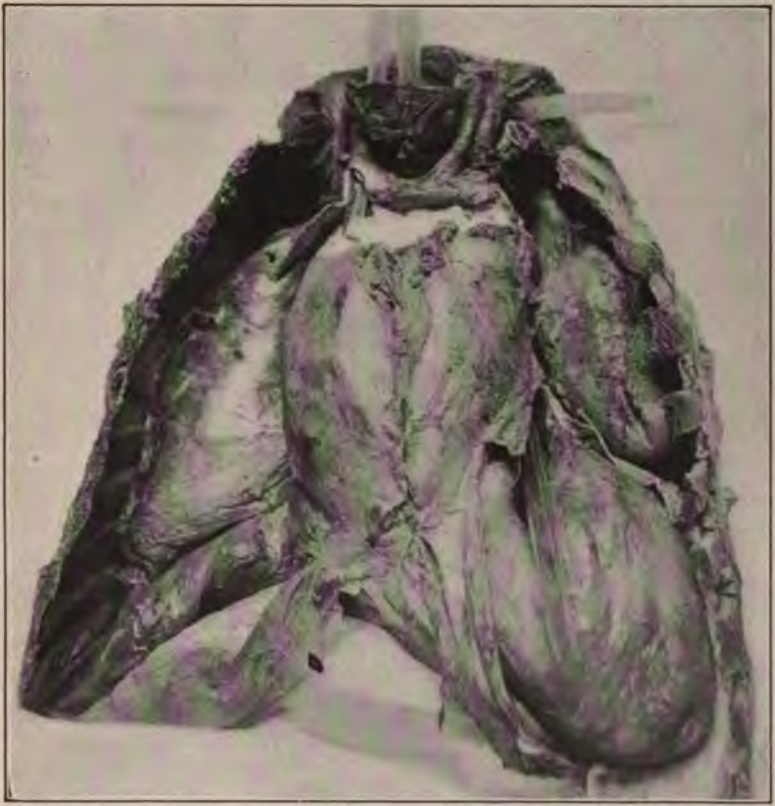


Fig. 93.—Lymphosarcoma of the mediastinum.

When a sarcoma starts primarily in the peribronchial lymph nodes it comes, in the course of time, to invade the glands at the root of the lung or in the mediastinum so that it is often very difficult to determine whether it be not secondary instead of primary (Fraenkel). In most cases of lymphosarcoma the new growth has originated in the mediastinal lymph glands and the clinical picture is in reality that of a mediastinal tumor. Such, at events, was the case in a male patient whom I had under observation a number of years ago. Secondary masses were discovered in the left lung *post mortem*, but the symptoms had been those of mechanical pressure.

Plates X and XI show beautifully secondary growths of the osteosarcomatous variety which completely studded both lungs and, during life, occasioned symptoms due to bronchial obstruction, namely, dyspnœa and chronic bronchitis. This case will be narrated in detail at another place.

Degeneration and softening of sarcomatous growths are not so frequent as in cancer. Virchow is said to have emphasized in particular the extreme rarity of such an occurrence in lymphosarcoma. These tumors lead to inflammatory adhesions between the lung and the costal pleura or other parts and, in particular, cause pleuritic exudates which may, as in a case once seen by me, completely obscure signs of the pulmonary disease. Some details of this case will also be narrated under the symptomatology.

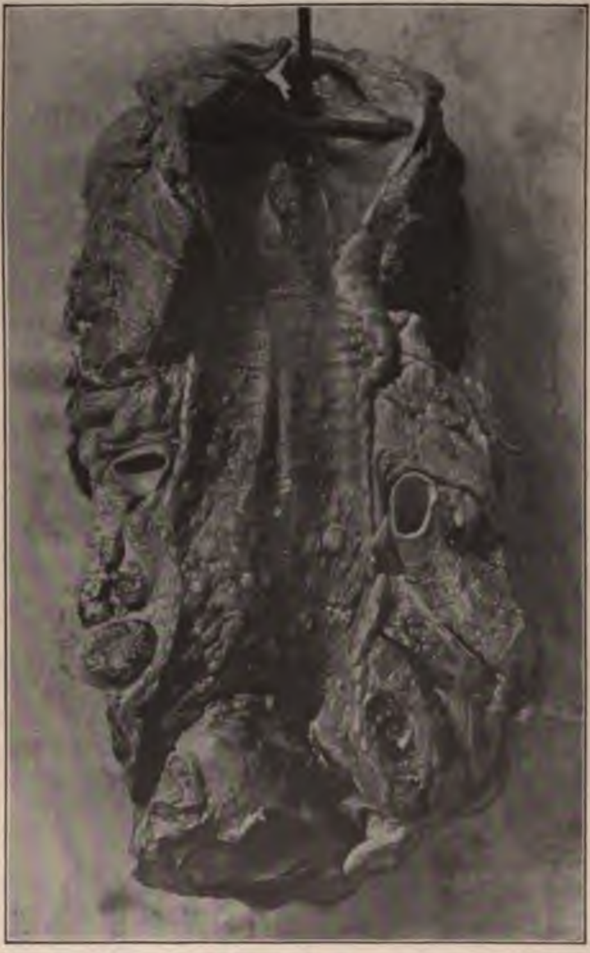
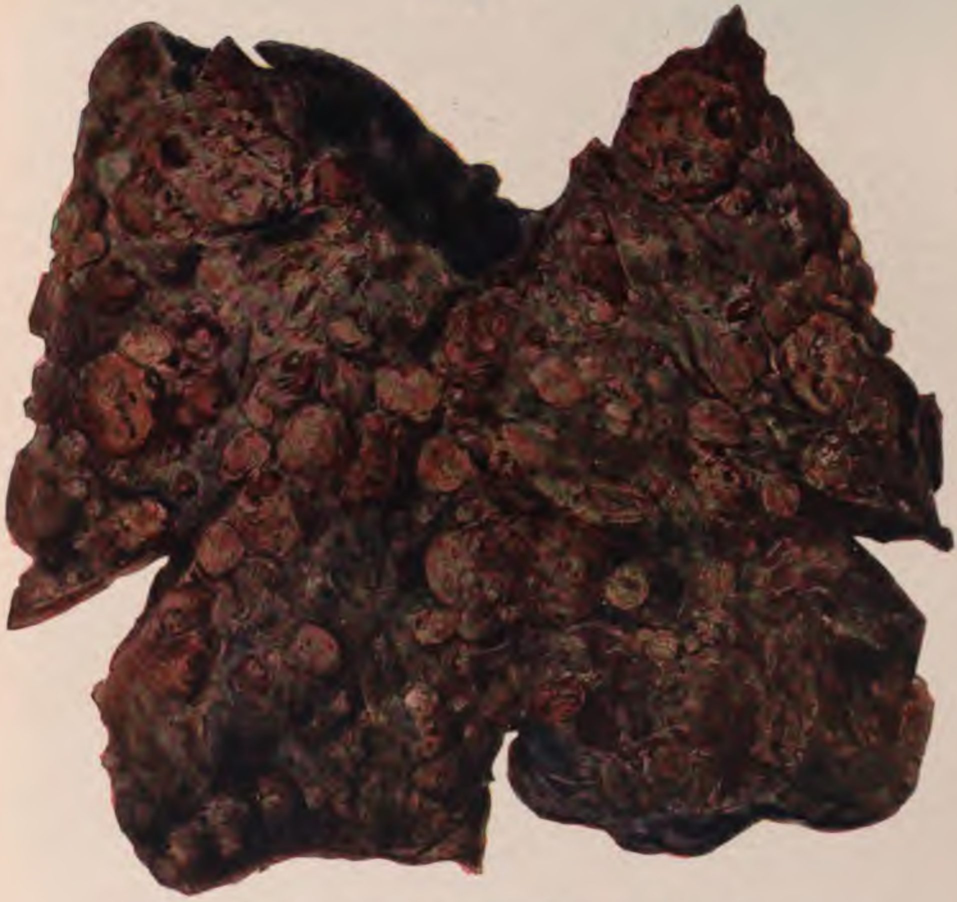


FIG. 94.—Sarcoma nodules in the trachea and bronchi and enlarged peribronchial lymph nodes.

Symptoms. — These present considerable diversity in kind as well as degree depending upon the seat and size of the neoplasm. The exact nature of the growth, whether carcinoma or sarcoma, can only be determined when the pulmonary disease is secondary to a primary tumor whose pathology is known. The symptoms occasioned by these two varieties do not differ essentially and therefore may be considered together. Neither is there any essential difference between the clinical picture produced by a primary growth and that by secondary disease of the lungs, excepting in so far as a primary lung tumor is apt to be single and of large

PLATE X



Cut Section of Lung Showing Multiple Nodules of Lymphosarcoma.

size, whereas, in the secondary form, there may be multiple masses of variable dimensions.

The disease of the lungs usually develops slowly, so insidiously in fact that the symptoms may not at first attract special attention. Exceptionally the patient may be able to date his sufferings from a definite time when he became conscious of some symptom due to mechanical pressure. Thus Aufrecht mentions the instance of a miller of sixty-five whose last illness appeared to commence abruptly with asthmatic breathing that came on directly after a hunting trip. Two weeks subsequently signs were discovered in the right side at the front base, dullness, enfeebled respiration and pectoriloquy, which aroused a suspicion of pleurisy with effusion. But aspiration yielded only a little blood.

Fraenkel narrates the case of a man in his fiftieth year who, for several months together, had expectorated blood without any exertion to cause it. At length, but four weeks prior to his death, dullness appeared in the right upper lobe and continued to increase. But such a history is very exceptional.

In the majority of cases the initial symptoms are cough, expectoration and dyspnœa, the last, in particular, varying much in intensity and character.

Cough is not often a troublesome feature and may be absent altogether. It depends upon the presence of secretions in the air-tubes and on their tenacity and may sometimes be paroxysmal. This symptom is likely to be more urgent when the growth compresses the trachea or a large bronchus and to be least marked when the body of the lung is invaded by cancer.

Expectoration is very apt to be absent or insignificant, according to West and Lenhartz, while other authors attach more importance to it and state that it may be of a sanguinolent character. Fraenkel's case cited above is one in point while Walsh states that less than a third of the cases of pulmonary cancer escape hæmorrhage entirely (West). Lenhartz also comments on the bloody appearance of the sputum which may vary from that of raspberry jelly to that of dark blood. Hæmoptysis was present in my case of osteosarcoma. The character of the sputa does not depend solely upon the collection and retention or the quality of the bronchial secretions, for if the growth softens and breaks down shreds or fragments of cancerous tissue may appear in the expectoration. Such, however, is not common.

In a case of osteosarcoma of mediastinum and lungs secondary to a tumor of the tibia, Huber discovered in the bloody expectoration fragments of the growth which were 5.5 cm. in length and 2.5 cm. in width and contained both small mononuclear and giant polynuclear cells, some of the latter having as many as 10, 20 and even 40 nuclei. Hampeln and Feldt are said to have made similar discoveries while Ehrich is reported to have found in the sputa of one case shreddy fragments of carcinoma (Fraenkel).

Apart from the occasional recognition of pieces of the neoplasm careful microscopic examination of the sputa may result in the detection of cellular elements of such unusual character as to be of diagnostic value. Thus Hampeln lays great stress on the presence of unpigmented polymorphous cells having distinct nuclei and nucleoli. They vary in size and may be seen as single specimens or in masses. Betschart reported the discovery of similar structures (Fig. 95) which, occurring in clumps of a diameter of 2 to 3 mm., were of a yellowish or brownish-yellow color and visible to the unaided eye.

In one of Fraenkel's cases A. Japha detected in the sputa, on careful microscopic examination, considerable numbers of epithelial cells which, having a layer-like arrangement and held in a stroma, presented an unmistakable picture of so-called cancer nests. Similar discovery was made in a second case. As pointed out by Wolf, the cells having a diagnostic significance are most likely to be found in cases which have not undergone ulceration, since this process is likely to destroy their characteristics.

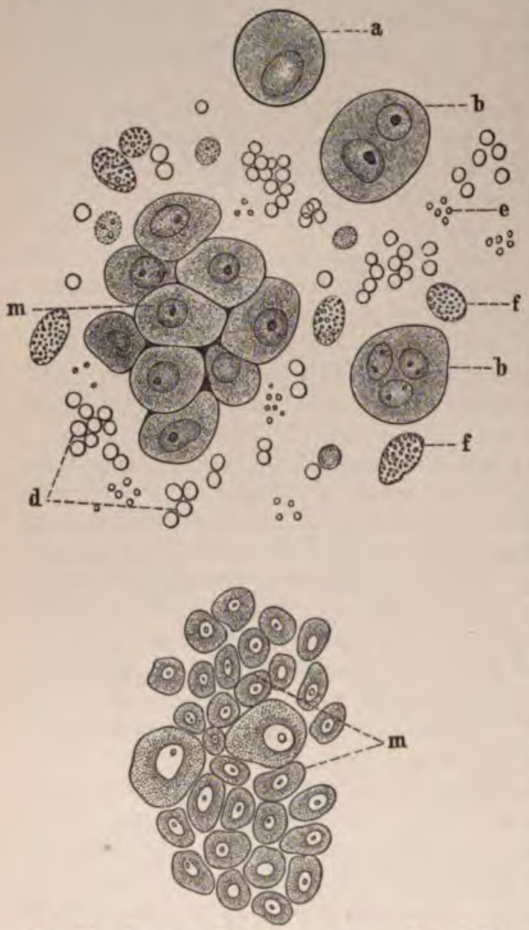
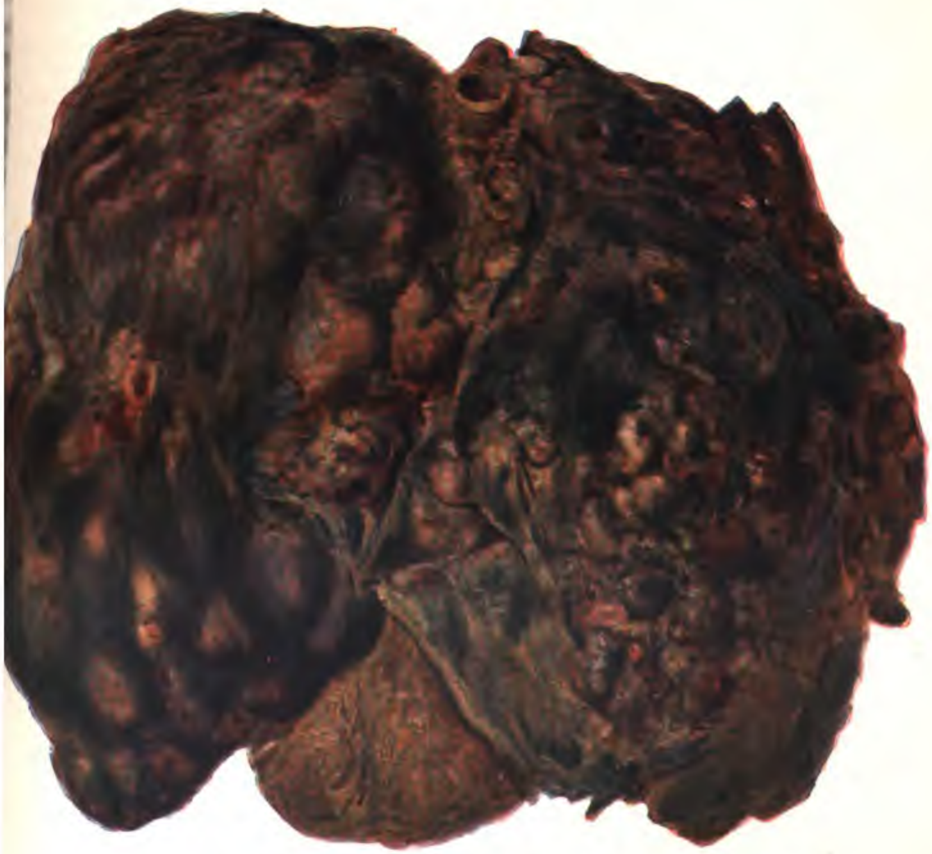


Fig. 95.—Tumor cells from sputum. (Fraenkel.)

Dyspnœa is very apt to be present in cases of pulmonary neoplasm but is determined by the size and situation of the tumor rather than by its special nature. Carcinoma of the body of the lung may, when primary and situated in the upper lobe, occasion great difficulty of breathing whereas at the base its pressure-effects are manifested in another manner. The explanation is to be found in the character of the structures compressed. At the summit of the chest there are important structures, as the trachea and large bronchi, the construction of which shuts off in corresponding measure the supply of air to the lungs and hence dyspnœa, even stridor, is the inevitable consequence.

A sarcoma may do the same thing. Thus a lymphosarcoma of the mediastinum and contiguous pulmonary parenchyma is very likely to produce dyspnœa amounting almost to strangulation, as in one of my cases that has been reported elsewhere. Situated at the base of the chest it interferes with respiration only indirectly through the exudate which its presence may call forth in the pleural cavity. On the other hand, multiple osteosarcomata are capable of producing almost unsupportable dyspnœa

PLATE XI



EXTERNAL SURFACE OF LUNGS FROM SAME CASE AS SHOWN IN PLATE X.

through the very considerable diminution of respiratory capacity resulting from their being distributed through the lungs in large numbers and everywhere compressing the bronchioles. The case from which Plates II and III were taken is one in point.

George Murray, a hostler, aged fifty-four, was seen by me in May, 1904, at the Cook County Infirmary, Chicago, where he was in the care of Dr. MacHugh, through whose kindness I was enabled to examine the patient and to obtain the colored representation of the lungs here shown. Family history was entirely negative but the personal anamnesis contained some highly interesting and important data.

As a young man he sustained a fracture of the right leg. Two years ago he was bitten by a horse on the left shoulder. The skin was not broken but badly bruised. This injury troubled him for about eight months during which time it was treated as rheumatism. About two months later, or ten months after the injury, a tumor began to form on the shoulder blade and he consulted a surgeon, who diagnosed it as sarcoma. He then amputated the shoulder, removing the clavicle and scapula entirely. The field of operation healed readily and for the following eight months he was free from any symptoms. About twelve months after the operation, and four months prior to the date of his admission to the Infirmary, he began to notice the present trouble.

The initial symptoms were insomnia, the man being unable to sleep until well on toward morning; slight hacking cough that had persisted and was more or less paroxysmal though never excessive; expectoration that at first was scanty and bloody, became at a later period more profuse and purulent, but at all times contained blood. Four months before admission he had quite a profuse hæmoptysis which was not repeated until the week before admission, when he had two free hæmorrhages.

Dyspnœa which was first noticed about two months ago, or twenty-two months after the injury and two months after the onset of pulmonary symptoms, at first slight, has gradually increased until at the present time there is an extreme grade of orthopnœa. At no time has he complained of pain, although he has a sensation of fullness or pressure in the chest, especially on the right side. There is no interference with deglutition but he has an aversion for food. The bowels, formerly regular, have for the past month been constipated. Loss of weight has been considerable in the last five months. There is no sweating, and the urinalysis is negative.

The general appearance was pale and sallow and the contour of the chest was peculiar owing to absence of the left shoulder. There was cyanosis of the face and hands and respirations were labored, the accessory muscles being called into play, the larynx moved up and down with each respiratory act and the intercostal spaces were seen to sink on inspiration. There were enlarged superficial glands in the cervical regions and on the abdomen to the left of the umbilicus as well as in the left midaxillary region between the fifth and sixth ribs. On the left side of the thorax, moreover, the superficial veins were dilated. The last two cervical and first two dorsal spines were unduly prominent. The pupils were small but equal and reacted to light. The supra- and infraclavicular regions were not depressed but distended and there was some pitting of the lower extremities.

The apex-beat appeared displaced downward and inward, the pulse was 132, respirations were 32 and temperature was 98° F. Vocal fremitus was decreased throughout both lungs, especially over the entire right lung and the left lower lobe, being of apparently normal strength only along the left border of the sternum. The lower margin of the liver was palpable one inch below the level of the umbilicus in the median line.

The apices yielded modified resonance on percussion, the right being almost dull. The left lung was flat up to the level of the fourth rib in front and above this there were areas of dullness up to the clavicle. The middle lobe of the right lung was absolutely dull from the fourth rib nearly to the upper margin of hepatic flatness and from the sternum to the anterior axillary line where the note became hyperresonant. A patch of dullness was determinable in the right axillary and another of the size of a small orange in the left axillary region. Percussion of the abdomen showed hepatic dullness to reach to the level of the umbilicus.

Auscultation showed decrease of vocal sounds in general and absence of voice-sounds over the flat area at the left base. Over the dull areas the breath-sounds were feeble and over the large flat area they were absent. In the intervening more or less resonant areas, the breath-sounds were obscured by moist râles. The auscultatory findings resembled those in the diffuse broncho-pneumonia of childhood or a general bronchitis with areas of atelectasis or pneumonia.

Microscopic examination of the sputa was negative as regards organisms or cells other than pus and blood cells. The expectoration resembled currant jelly in color in consequence of its containing blood. A lymph node of the size of a hazelnut was removed from near the umbilicus and, on section, showed numerous multinuclear cells in rather close connection with the connective-tissue stroma, and appeared to be sarcomatous. The pleural fluid removed by means of a small hypodermic syringe showed a few endothelial cells and some red blood cells.

Post mortem, May 19th, and fourteen days after admission. With exception of the superficial glands already mentioned and atrophy of the chest muscles on the left side from disuse, there were no changes worthy of note outside the thorax. Right lung.—This comes over to the median line and is very firmly adherent to the chest-wall. The pleural cavity is almost obliterated on this side by numerous old adhesions and the pleura is torn in the attempt to remove the lung. There are numerous sacculated pockets of fluid throughout but particularly along the spinal column. This lung measures 10 by 6 by 4 inches. There are numerous growths scattered throughout the lung which have a general spherical form and shell out easily from the surrounding tissue. Left lung.—This appears normal in position and is free from adhesions except at the lower border of the upper lobe and in the midaxillary line. There are about 10 ounces of bloody fluid in the pleural cavity. The bronchial lymph glands are enlarged. This lung is very firm, large and heavy, filling the cavity of the thorax completely. It measures

12 by 7 by 4½ inches. The interlobar markings are almost obliterated. The upper lobe of this lung crepitates and the lung is completely filled with yellowish tumor masses varying in size from a pin's head to a hen's egg. These growths present various stages of consistency, the smaller and apparently more recent being soft and succulent while the larger and older are firm and hard and have a gritty consistency on section. The masses in both lungs are similar and those near the surface of the organ fall out of the surrounding substance wherever the pleura is torn. On cross section of the lungs they are both found completely infiltrated with the nodular growths. The color of the lungs is normal except that it is slightly more pigmented than usual. With exception of the hypertrophy and dilatation to be expected and the changes of chronic myocarditis the heart is negative and the pericardium presents nothing worthy of note.

Pain may or may not be a feature of malignant disease of the lungs, depending upon the circumstances of the individual case. The involvement of the pleura is likely to be attended with more or less pain, especially if it leads to inflammation and exudation. If a massive tumor occupy the upper part of the thorax and encroach upon the parietes, it will almost inevitably occasion suffering in consequence of pressure on the intercostal nerves or on the branches of the brachial plexus. In the latter event the pain may extend down the arm. In fact the pain is complained of only when the growth encroaches on the external parts, the same as in aneurysm, for it is very doubtful if the pulmonary parenchyma itself is sensitive in the way of actual pain.

In the Winter of 1902 I saw in consultation with Dr. I. C. Anker of Chicago a lady of fifty who was a great sufferer from pain in the left side of the chest and from cough which was frequent and dry. My first examination was negative or practically so. There were rather indefinite signs pointing to some mischief in the left lung. No positive diagnosis was made by me but the case was suspected to be one of tuberculosis. During the succeeding weeks the patient's symptoms increased and she had daily recourse to 5-grain doses of acetanilid for relief from her pain. In addition she lost moderately in strength and weight.

In the fore part of March I again saw her and then had no difficulty in recognizing the presence of fluid in the left pleural cavity. This confirmed me in the opinion that her disease was tuberculosis, but I was at a loss to account for the persistence and severity of the pain, since this is not usual in tuberculous pleurisy. Her dyspnœa was such that I recommended aspiration for its relief.

Accordingly, Dr. Bayard Holmes was summoned and withdrew about a quart of bloody serum. This afforded a temporary improvement in the symptoms; but before long the fluid reaccumulated and another tapping was done. The fluid obtained this time had a specific gravity of 1.025 and was rich in albumin. It was alkaline in reaction and contained lymphocytes and erythrocytes to which last it owed its bloody color. The patient's blood showed a leucocytosis of 15000, a fact which should have led to the opinion that the condition was not tuberculous. I was not seeing the woman at that time and have since learned these facts from the report of the case made by Dr. Holmes's assistant, Dr. A. Bremken. The patient died in August, 1902, and the autopsy was performed by Dr. Anker. I condense the report given in Dr. Bremken's paper as follows:

"The left pleural cavity contained about 3 pints of thick, bloody, brownish-red fluid; no adhesions were present. The lung was of about the size of two hen's eggs; the upper lobe being very friable and bloody, the lower portion of the lung being hard to touch and containing considerable blood; the parietal pleura was smooth and glistening; no enlarged lymph glands in the mediastinum. Right lung.—The parietal and visceral pleura were smooth and glistening and there were no adhesions; no enlarged lymph glands were found; the lung was about one-third the normal size and small white nodules were seen in the visceral pleura. The heart's apex was in the mid-sternal line and there were no pericardial adhesions. The aorta was bound down by adhesions and was atheromatous. The diaphragm was thickened throughout, especially on the left, and the thickening was plainly due to invasion by the neoplasm. The abdominal organs displayed no signs of malignant disease.

"Sections from an area of consolidation including the margin of the lung show irregular islands of epithelial cells. These cells are separated from each other by a connective-tissue stroma, rich in well-staining connective-tissue nuclei. Fingerlike projections of the stroma are seen everywhere. The arrangement of the cells in the groups is always more or less characteristic. The outer layer of cells or those in contact with the stroma are cylindrical, while the others or those lying internal to this layer are more or less irregular in outline, and they vary in size. As we reach the periphery of the tumor an entirely different picture presents itself. Here it attains the type of a cystoadenocarcinoma" (Fig. 96).

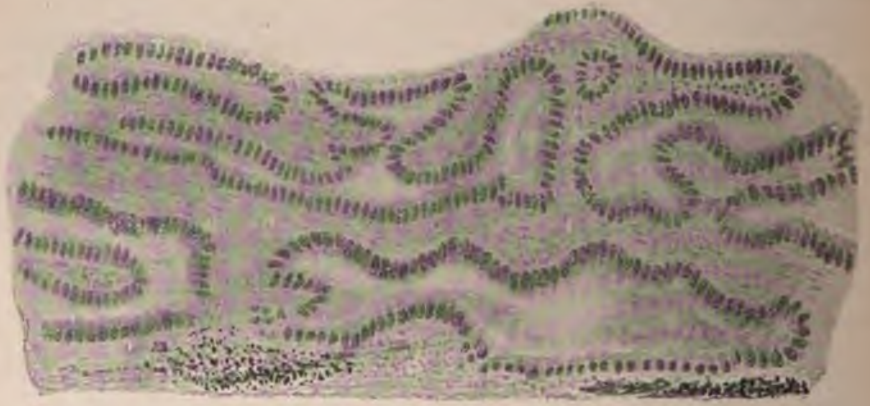


FIG. 96.—Microscopic section of tumor in case of carcinoma of lung. (Bremken.)

The case was probably one of primary carcinoma of the lung and illustrates the diagnostic importance that should be attached to severe and persistent pain before distinct evidence of pleuritic involvement.

Symptoms due to mechanical pressure are variable in frequency and severity depending upon the conditions residing in each case. A primary neoplasm whether sarcoma or carcinoma occupying the upper portion of the chest is almost certain to press upon some of the structures in this region, in particular upon some of the large blood-vessels. Accordingly, one of the not uncommon features of such a case is *dilatation of the superficial veins.*

This is especially apt to result from pressure upon the vena cava superior or one of its large tributary branches in the anterior superior mediastinum. In such an event the veins are likely to be dilated on the side of the neck and chest, and in the arm corresponding to the situation of the growth. Such a unilateral dilatation of the veins is one of the diagnostic features on which much stress is laid by some clinicians. It was this finding that enabled a certain physician of my acquaintance many years ago to make a correct diagnosis in a case of lung cancer which I had also seen and failed to recognize as one of pulmonary cancer on account of the pleuritic effusion present and for which the lady was tapped.

In this particular case it may be stated that there was a history of previous operation for some sort of a growth on the wall of the chest. Its precise location I do not now recall, but it should have created in my mind a suspicion of the nature of the intrathoracic disease. It is also most interesting and significant that after my examination of the case the carcinomatous tissue appeared externally through one or more of the sites of previous paracentesis. So far as I know no autopsy was had in this case, and it is probable that the neoplasm invaded both the pleura and parenchyma.

The dilatation of the superficial and ordinarily invisible venules of which I have spoken is not at all a certain sign of malignant disease within the thorax but merely an indication of some condition that interferes with the circulation within the anterior mediastinum. As such, however, it should be carefully sought for and may be found not alone on the upper surface of the chest but at the base where there may be a plexus of veins traversing the anterior wall in a line corresponding to the level of the diaphragm, or a perpendicular cord about the size of a goose quill passing from the ensiform to the umbilicus and caused by distention of the superficial epigastric vein.

In connection with this state of the veins there may be œdema of the skin of the neck and chest or of an arm as a still further proof of mechanical interference with the circulation in the parts drained by the dilated and partially thrombosed veins. There may, in some cases, be an inequality of the pulses on the two halves of the neck and in the arms (Fraenkel).

Pressure effects may be shown still further by paralysis of the vocal cords and consequent hoarseness or aphonia, by localized bulging of some region at apex or base and consequent want of conformity in the two halves of the thorax, by localized pulsation in an abnormal situation and by tracheal tug.

That destructive pressure upon the recurrent laryngeal and localized enlargement of the thorax may be produced will not strike one as remarkable, but that pulsation and tracheal tug may be produced by a solid tumor as well as by an intrathoracic aneurysm will surprise many.

Nevertheless, Fraenkel is authority for the statement that tracheal tug (the Oliver-Catarelli sign) may be produced when, in consequence of associated involvement of the mediastinal lymph nodes, a tumor mass is situated between the inferior surface of the aortic arch and the left main bronchus in such a manner as to communicate the pulsation of the vessel to the air-tube. His former assistant, M. Auerbach, discovered such a state of things in two of Fraenkel's cases. The author states also that an adhesion between a cancer and the side of the trachea might occasion the same clinical phenomenon.

Localized pulsation from a solid growth is systolic in time but lacks all semblance of the expansile pulsation sometimes apparent in aneurysm of the thoracic aorta. In my experience it is not pronounced and may be so vague as to create considerable hesitation and even doubt in the mind of the observer as to its real existence. The detection of such pulsation is better accomplished by the eye than by the hand and requires minute inspection in a good light.

In a word, any sort of pressure effects may be present in some cases of pulmonary neoplasm. Thus compression of filaments of nerves belonging to the sympathetic system may occasion inequality of the pupils and localized or unilateral sweating. It is probably safe to say that when pressure symptoms are marked there is either a massive primary lung tumor or the malignant disease as a lymphosarcoma, invades also the mediastinum.

Glandular enlargement in the cervical region, just above the clavicle or in the axilla, is another objective symptom that may give a clew to the presence of a neoplasm, particularly lymphosarcoma. Enlargement of the lymph nodes in the neck is not invariable but, when present, forms an important link in the chain of diagnostic data. In one case which I saw a couple of times a number of years ago, that of a druggist of about fifty, who suffered from signs and symptoms of intrathoracic pressure, there appeared above the inner third of the left collar bone a mass the size of half a butternut, or the terminal phalanx of a man's thumb. This tumor, which may or may not have been an enlarged and indurated lymph gland, was intensely hard and slightly sensitive to pressure. There were no other palpable nodes that I now recall. The precise nature of this man's pulmonary disease could not be ascertained as an autopsy was denied.

Pulmonary neoplasms, even when primary and especially when secondary, rarely confine their inroads to the parenchyma but occasion symptoms referable to involvement of the pleural cavity. In such instances the signs of pleuritis with effusion may wholly obscure the disease of the lung proper. The following case narrated from memory is one in point.

In March, 1894, I was called to a town in the interior of Illinois to see a young man, the brother of a colleague, who stated that his brother had been ill with some form of chest disease for several months. The man was very pale and thin, and confined to bed. He was running a moderate daily temperature of a degree or two above normal and had a dry, not very troublesome cough. The pulse was feeble and considerably accelerated out of proportion to the pyrexia. He had been losing strength and weight more rapidly in the few weeks immediately prior to my visit.

A careful examination of the chest resulted in the discovery of nothing more than signs of a moderately extensive pleuritic effusion in the right half of the chest, dullness to slightly above the nipple, diminished fremitus and breath-sounds. In consequence of the moderate fever and the absence of signs of apical disease the opinion was expressed that the whole trouble lay in the pleural cavity and an exploratory puncture was advised for the purpose of ascertaining the nature of the exudate. Accordingly this was done without delay and resulted in the withdrawal of a clear serous fluid having all the appearance of an ordinary sero-fibrinous effusion. The case was pronounced tuberculous, therefore, and an unfavorable prognosis was given.

The patient was not seen again and death occurred not many days thereafter. Details of the autopsy were not transmitted to me but I received the rather curt statement from the brother that the disease was sarcoma of the lung. I had then and still have the impression that the doctor thought but poorly of my diagnostic ability inasmuch as I had failed to recognize the exact pathological condition.

Without wishing to mitigate my offense in that instance it might be stated that had the exudate been bloody it might have aroused a suspicion of the nature of the pleurisy. The man was not quite thirty years of age, as I now recall.

Fever may or may not be present in this class of cases. Fraenkel states that it was a feature of nineteen out of his thirty-five instances of malignant pulmonary disease and was of an irregularly intermittent type. Its cause was various, depending upon the absorption either of the products of destruction of the tumor or of secretions retained within the obstructed and inflamed bronchi. In the case just mentioned neither of these causes existed, in my opinion, and I am inclined to attribute the pyrexia to the pleuritis. There is, therefore, nothing in the fact of the neoplasm *per se* to cause an elevation of temperature.

Dysphagia is also a possible feature and may be the result of pressure by the tumor itself or by swollen and degenerated peritracheal or mediastinal glands. *Cachexia* is not likely to be a conspicuous symptom and in the cases coming under my observation it has not been pronounced. According to Herrmann quoted by Fraenkel it develops more slowly and is never so marked as in cases of gastric or intestinal cancer. Hampeln, who also has studied the marasmus of malignant disease, says it is a feature of neoplasm in any of the viscera but in the case of pulmonary cancer presents nothing unusual, being an expression of anæmia and being especially pronounced in cases in which it is combined with poverty and old age.

Course and Termination.—The course of malignant disease of the lung is as varied and uncertain as in cancer of other organs. According to Lenhartz, it may extend over a period of a few months to two years, the most of his cases having fallen within these limits and yet, as will be shown, the clinical course may be embraced within a few days or weeks. In some cases the disease is latent, running its course without giving rise to any symptoms by which its existence can be suspected, while in others obscured by the symptoms of chronic bronchitis and emphysema, cardiac disease or old age.

The termination is usually through exhaustion but may be in consequence of heart failure or suffocation. Exceptionally, the course of the disease is abruptly terminated by hæmorrhage or by some inflammatory or gangrenous process that on *post-mortem* examination is found due to the growth the presence of which may or may not have been surmised. Lenhartz cites the case of Paessler's in which a fifty-two-year-old man who had shown no other symptom than hoarseness and trivial cough was suddenly seized with a profuse hæmoptysis which, after two slight repetitions on the succeeding day, was followed by a third hæmorrhage so profuse that death occurred on that same day. Upon autopsy it was discovered that a cancer whose presence had been wholly unsuspected had perforated a main branch of the pulmonary artery.

Another of Paessler's cases cited by Fraenkel also ended abruptly without a suspicion of the underlying pathological process. A healthy man of fifty years became ill suddenly with rigor, fever and pain in the chest. Four weeks later these symptoms were repeated and there was discovered a small patch of dullness in the lower portion of the left scapular region. The expectoration soon thereafter became profuse, putrid and of the other characters of gangrenous sputum. Death occurred at the end of four weeks and at the necropsy there was found a cancer in the interior of the left lower lobe which had caused stenosis of one of the bronchi. Peripheral to the stenosis were numerous bronchiectatic cavities and areas of bronchopneumonia that were undergoing gangrene.

Such a sudden and unexpected termination of an insidiously developing pulmonary neoplasm also occurred in Fraenkel's practice. A physician of forty in hitherto robust health became abruptly ill with an obscure inflammatory affection of the right lower lobe characterized by dullness, feeble bronchial breathing and coarse bubbling râles. An exploratory puncture was negative. Expectoration became horribly offensive and toward the close of the second week of his illness the patient lapsed into unconsciousness and died. At the autopsy the right lung was very voluminous, especially the lower lobe which was nearly devoid of air and of doughy consistence. On section there poured forth an enormous amount of puriform material and the parenchyma was soft and easily penetrated by the finger. In various places were cavities surrounded by broken-down tissue. The bronchi were somewhat dilated and filled with a similar secretion. In the main bronchus of the lower lobe was a mushroomlike growth which, springing from the posterior wall, nearly obstructed the lumen of the tube. The tumor was medullary and very soft and stood forth conspicuously above the dark-red mucosa. On section it was found to be connected with a similar cherry-sized cancerous nodule that seemed surrounded by a capsule which, situated at the hilus of the lung, was evidently a lymph gland.

The partial obstruction of the bronchus occasioned by the growth had led to retention of secretions which, under the action of bacteria, became putrescent and thus excited the inflammatory gangrenous process of which the man died. Another case, that of O. Koerner's, illustrates another phase of the results likely to follow a pulmonary neoplasm, namely occlusion of a main bronchus and atelectasis of the entire lung.

The patient, who was sixty-four years of age and had manifested symptoms referable to an old kidney and heart affection, was suffering from a fibrinous bronchitis of two months' duration when one day Koerner discovered a slight flattening of the right front chest-wall between the second and fourth ribs. Over this area the percussion-note was impaired and the inspirium was sharpened. The retraction of the chest-wall and the dullness increased in time but the breath-sounds disappeared, returning again later on, as bronchial though feeble. Gradually the retraction of the thorax reached an extraordinary degree. The percussion-note in right front became intensely flat, fremitus and bronchophony were wanting, the left lung expanded until it reached to the right of the sternum. At the right back the note was impaired but not actually dull.

At the autopsy the right lung was totally retracted and devoid of air but not inflamed. The lower portion of the trachea was surrounded by a mass of cancerous glands. On the anterior wall of the lower third of the tube was a soft tumor of the size of a hazelnut. A little farther down was a second growth which, 5 cm. long and 2 cm. broad, obstructed the right main bronchus, while in the branch leading to the lower lobe were similar growths as well as a diffused cancerous infiltration which occupied the lumen of the tube. All the tumor masses had penetrated the bronchial wall and were connected with and probably originated in carcinomatous glands on the exterior of the tube. During life a diagnosis had already been made of cancer at the root of the lung.

Involvement of the Pleura.—Another condition which under certain circumstances may prove a serious complication but in any event is apt to embarrass diagnosis, is the invasion of the pleura by the lung tumor. In most cases this is manifested as a more or less acutely developing exudation. Exploratory puncture shows this exudate to be quite various, more commonly hæmorrhagic, but sometimes serous and macroscopically like that of an ordinary pleurisy, as in my case of sarcoma already narrated. In fact this sanguineous fluid may be so dark as to resemble pure venous blood (Fraenkel). In exceptional instances the exudate may have the appearance of a fatty emulsion, due, as Quincke has shown, to a fatty degeneration of the cellular elements. Now and then cancer particles may be discovered in the fluid.

Pneumothorax is a complication which, according to Fraenkel, is produced in rare cases either by degeneration of the growth in the lung and consequent establishment of a fistula between lung and pleural cavity or by laceration of the pulmonary parenchyma at some point along the line of puncture between the lung and the circumscribed pleural adhesions. Under such circumstances some unusual respiratory effort might easily cause a tear in the lung and the sudden development of the symptoms and signs of escape of air into the pleural cavity.

Physical Signs. Inspection.—Whether or not malignant disease of the lungs occasions signs of its presence apparent on inspection depends upon the extent and location of the growth. A primary tumor, therefore, is more likely to occasion visible signs than are smaller secondary masses even though they be multiple. Moreover such ocular evidences of disease as present themselves are often the result of an associated mediastinal growth or of a secondary pleuritis with effusion.

The most common change discernible is some modification of the normal configuration of the thorax showing itself either as a localized bulging of the chest-wall or a uniform enlargement of the side. As might be expected such a prominence is more readily produced when the neoplasm occupies the upper, not the lower part of the lung. A pleural exudate however will declare itself by increased expansion at the base. See Pleurisy with Effusion.

That the change is not always shown by enlargement of the side, either local or general, is shown by Koerner's case narrated above in which flattening and retraction of the corresponding half of the thorax occurred. Pressure effects may also be visible in the way of venous dilatation, œdema of the face, neck, chest or arm, enlarged lymph nodes, etc. Localized pulsation though not so likely to occur as in cases of thoracic aneurysm should be carefully looked for, but to be discernible necessitates inspection in a good light. There may be more or less pallor and emaciation or the patient may present a well-marked cancerous cachexia. But this is not so likely as in malignant tumors of other viscera.

Palpation.—This is not likely to afford much additional information unless it happen that the growth interferes with the transmission of vocal fremitus in some conspicuous and anomalous manner. Thus fremitus may be absent over a prominent or retracted area at the summit of the chest where the detection of dullness would lead one to expect increase rather than decrease of pectoral vibration. Palpation is also likely to assist in the detection of a pleuritic exudate, displacement of the heart, inequality of the pulses on the two sides of the body, œdema, and localized bulging. On the whole, however, it is of chief value in the study of peculiarities of pectoral fremitus over limited regions or on one side of the chest, since the conduction of vibration is interfered with by atelectasis or the presence of a tumor mass obstructing a bronchus or situated sufficiently close to the periphery of the lung.

Percussion.—This may or may not be of aid in the recognition of pulmonary neoplasm. If the growth be small and deeply situated it may occasion no appreciable alteration of pulmonary resonance. On the other hand, a primary tumor of considerable size and located in the upper part of the chest will probably give rise to dullness more or less marked and of limited extent, or multiple nodules may occasion many areas of dullness scattered in various parts as in my case of osteosarcoma. If the lung cancer be associated with cancerous mediastinal glands or a primary tumor of the mediastinum, there is likely to be dullness in the upper sternal region. This dullness, moreover, is apt to be of irregular outline and to encroach upon one or the other infraclavicular regions. As the disease advances it is likely to be evinced by progressive increase in the area of dullness. Cancer at the root of the lung is apt to produce dullness in one of the interscapular regions.

A remarkable phenomenon elicited by percussion in exceptional cases and one on which Fraenkel lays particular stress, is a transition from tympany to dullness, and again from dullness to tympany over a lobe or a limited region. This change of note from time to time in the progress of the disease was observed by him in the following case. September 23d a fifty-two-year-old laborer in a condition of comparatively good strength and nutrition was admitted to the hospital and stated that for two years he had suffered from stitchlike pain in the left half of the chest, cough and at times a slightly bloody expectoration. He presented moderate dullness, bronchial breathing and copious coarse râles over the left lower lobe. He ran a moderate and irregular fever and gradually developed a somewhat indurated and enlarged inguinal gland on the left side. Toward the end of October the left upper lobe became dull-tympanitic which note, after several days, became so dull that the upper border of cardiac dullness could not be distinguished therefrom. The breathing was feebly bronchial in front, scarcely audible behind and accompanied by a few râles. Repeated exploratory punctures in several places were negative. An X-ray examination showed almost complete consolidation of the left lung. About the middle of November the dullness began to lessen and tympany gradually returned over the left upper lobe. At the same time slight retraction of the lung was observed. In the beginning of December the note over the left upper lobe was fully sonorous and no longer tympanitic except over an area 2 inches in diameter in the second interspace. It again gradually lost in resonance, became tympanitic and by the end of the month passed into complete dullness which once more in February became slightly tympanitic for a short time. The breathing remained as before, feeble and bronchial without noteworthy râles. The retraction of the left chest in front increased. The enlarged gland in the inguinal region was removed and examined and proved to be carcinomatous.

The autopsy disclosed universal adhesion of the left pleural surfaces, the union being so firm that in removal of the lung the costal membrane came away with the lung. The entire left lung was devoid of air and the upper lobe of cartilaginous hardness. The left main bronchus directly below the bifurcation of the trachea was nearly occluded by a nodular mass of reddish-white color and soft consistency which tumor extended into the branch leading to the lower lobe. The branch leading to the upper lobe was invaded by the growth mainly in its first portion. On section the entire lung was infiltrated with numerous yellowish-white medullary nodules of the size of a lentil to a pea which were partly confluent and partly separated by the thickened grayish-white interlobular septa. At the hilus of the lung was a medullary tumor of the size of a man's fist which had invaded the parenchyma. The bronchial glands at the bifurcation presented the same white medullary consistency. The pleural adhesions over the upper lobe were 2 cm. thick. The microscope showed the growth to be a cylindrical-celled carcinoma.

A somewhat similar observation was reported by Grünwald. The patient suffered from a left-sided carcinoma of the lung which, however, caused bronchial stenosis by pressure and not perforation of the bronchi. During life Grünwald observed that when the patient lay in the dorsal decubitus the area of dullness stretching from the second rib to the upper margin of cardiac dullness became tympanitic. Over this area the breathing was bronchial. The necropsy disclosed extensive disease of the mediastinal glands and in the interior of the lung a soft mass of the size of a fist. Grünwald believed that the change in the percussion-note was best explained by assuming a modification in pressure according to posture.

It may here be stated that this alteration from tympany to dullness and the reverse is quite different from the *tympanisme thoracique* which Woillez declared sometimes preceded dullness and which is caused by relaxation of the lung; this he thought might be of assistance, therefore, in the diagnosis of lung cancer. But such a tympanitic note may be observed transiently in other conditions, as in the commencement of a pneumonic exudate and of an intrapleural effusion. It has not, therefore, anything like the significance of the phenomenon noted by Fraenkel and Grünwald.

Another phenomenon associated with impaired resonance and best detected on percussion is a *sense of greatly increased resistance.* This may impart to the entire lung a feeling of its being firm and resisting like a board or a stone, or this sensation may be perceived only in certain areas. Whereas this sense of increased resistance is not necessarily present in cases of lung tumor nor by any means limited to this disease, still if perceptible in connection with other suspicious signs it would have considerable weight.

Auscultation.—There is no constant or invariable auscultatory phenomenon attributable to neoplasm within the lung. If the growth leads to compression of a bronchus of considerable size, or to occlusion by its perforation of and growth within the tube, the breath-sounds over the area supplied by the bronchus will be weakened and broncho-vesicular or bronchial. If complete atelectasis develops over a region sufficiently close to the surface, the respiratory murmur will be abolished. The same results when the tumor-mass *per se* occasions dullness in any given zone. Over such an area the voice-sounds and vocal fremitus are weakened. Hence, in a suspected case of lung cancer such a localized diminution of voice- and breath-sounds possesses considerable value in the recognition of the nature of the case. The presence or absence of adventitious sounds is determined by some secondary condition as bronchitis, etc. The absence of vascular bruit is of importance in cases presenting pressure effects, since although a murmur may be wanting in aneurysm its absence speaks for rather than against solid tumor.

Diagnosis.—From a diagnostic standpoint malignant tumors of the lung may be divided into three classes; namely, (1) a relatively small number of cases in which the disease can be recognized by particles of the growth in the sputum or in the pleural exudate; (2) those cases forming the largest proportion in which diagnosis is possible or only presumable from the physical signs strictly speaking; and (3) also a relatively large number of cases the real nature of which is concealed beneath symptoms and signs of some associated or secondary disease.

The first group requires few remarks additional to those that have been made already concerning the constituents of sputa or pleuritic effusions in the part of this chapter devoted to symptoms. *Cyto-diagnosis* has already yielded and is likely to yield results that will amply repay the worker in this field. Physicians are too apt to content themselves with examining specimens of expectoration for bacteria and to report or receive a report as negative any sputum that fails to show these. In every suspicious case the sputa should be often and carefully examined with a view to the possible discovery of fragments of a growth or of the polymorphous unpigmented polynuclear cells reported by Hampeln and Betschart.

Krönig's procedure of aspirating the lung itself by means of a fine hypodermic needle might also yield fruitful results in the way of cancer cells but is not devoid of danger. If the growth be in a state of degeneration and softening there is risk of hæmorrhage, but if the tumor should chance to be lymphosarcoma no serious result would ensue.

The fluid withdrawn from the pleural cavity in any case suspected to be one of malignant disease should be made the object of especial study. The detection of cancer particles either microscopic, or, if united in cell-clusters of sufficient size to be discernible as globular masses of about the size of a pin's head, would determine the diagnosis. Should the exudate be milky in appearance and contain small masses composed of fat-granules, or should it be composed of almost pure blood, it would in either event strongly suggest malignant growth. Unfortunately a bloody, fatty or purulent pleural fluid may be found in tuberculous disease, but should there be symptoms or signs referable to intrathoracic pressure the nature of the fluid would make very strongly for neoplasm.

I have seen a case in which an exploratory puncture of the right half of the chest resulted in the withdrawal of pure blood, and yet the disease, supposed to be a solid tumor, proved to be a massive aortic aneurysm. The details may be given briefly as follows: A man was presented before the Chicago Medical Society showing symptoms and signs of intrathoracic pressure, dyspnœa, distention of the right half of the chest, unilateral dilatation of veins on the surface of the neck and chest, flatness over the entire right chest and absence of breath- and voice-sounds on the same side excepting at the extreme upper and posterior part, no heart tones or bruits in the dull area, no pulsation. Upon such data the diagnosis was very dubious, but preponderance of evidence seemed in favor of solid growth as against aneurysm. Nevertheless the autopsy disclosed an enormous aneurysmal sac that completely filled the right half of the thorax and had compressed the lung upward and backward.

The second class of cases in which diagnosis is possible or only inferential constitutes by far the largest number. In these it is the study of history, the symptoms and the local findings which must be relied on for diagnosis. The history of previous operation for malignant disease of mamma, stomach, uterus or bone, in the presence of chest symptoms coming on some months subsequently, would enable one to come to a diagnosis with considerable certainty. The same holds true of the discovery of cancer in some other part coincident with pulmonary disease.

Circumscribed or unilateral prominence of the chest-wall is a sign of great diagnostic value, but its importance depends largely upon its situation.-

If the bulging is at the base, is associated with dullness, feeble bronchial or absent breathing and diminished fremitus, and if it is found by puncture or otherwise not due to intrapleural effusion, then it tells very strongly for malignant tumor and probably a primary one of the parenchyma. If, on the other hand, the distention of the chest be in the upper part and in direct relation to the anterior mediastinum it may indicate a variety of conditions, chief among which is aneurysm. Moreover, if there be a neoplasm it may be of the mediastinum or of a main bronchus and not the lung.

Further data that point to pulmonary growth are dullness, more or less defined, over the bulging area with diminished bronchial or absent respiratory murmur and decreased or abolished fremitus, no pulsation, no heart-tones and no bruit over said area. Should there be, perchance, an intrapleural fluid the condition is probably a solid growth.

This conclusion would be confirmed if, after a time, appreciable increase in the size of the area could be demonstrated, or if the dullness already shows or should assume an irregular outline.

Additional evidence as to its nature would be found in the detection of an enlarged isolated gland in the supraclavicular fossa, in the axilla, on the side of the chest or on the wall of the abdomen, as in the case of osteosarcoma which has been narrated. Nevertheless, valuable as the discovery of such a gland may be, it is not conclusive for, although tuberculous cervical glands are generally multiple they are not always so. In the Spring of 1904 I saw in consultation a lady of about fifty who had signs of pleuritic effusion on the right side. This fluid had made its appearance rather abruptly some weeks earlier and had accumulated rapidly after each tapping. It was serous in appearance. The lady complained sorely of pain in that same side. Her temperature was practically normal. On the whole, I was inclined to regard it as a tuberculous process but discovered a single lymph node of the size of a cherry directly above the middle third of the clavicle. No other glands could be palpated. Thereupon I expressed the opinion that the disease was probably malignant. When last I heard from this patient nearly a year subsequent to my examination her general health was improved and the gland in question had disappeared. The conclusion seems inevitable that it could not have been malignant.

Pulsation of the bulging area or over the supposed site of cancer as well as tracheal tug makes against a neoplasm, notwithstanding Fraenkel's assertion that they both may be occasioned by a new growth. Probabilities must always be taken into account in making up a diagnosis and certainly the probabilities are against these two phenomena being due to malignant disease. Nevertheless, the possibility of their occurrence in this class of cases must not be forgotten.

If localized pulsation is produced it is rarely pronounced and is not at all expansile, being merely the visible manifestation of an impulse imparted to the tumor by the expansion of some blood-vessel, as the aorta. A tracheal tugging due to a mass of cancerous glands between the aortic arch and left main bronchus would, at the best, be but a slight downward pull and not the vigorous jerk felt in aneurysm.

The sudden or gradual alternation of tympany and dullness observed by Grünwald and Fraenkel is so rare as to be practically of no assistance in arriving at a diagnosis. Should the phenomenon be observed, however, it must, in the light of such testimony, weigh strongly in favor of malignant disease of such nature and situation as to lead to a partial or complete obstruction of a bronchial tube.

The occurrence of moderate and irregular fever, together with pressure-symptoms, points toward malignant disease. Its absence in nowise tells against it. Age is a factor of considerable importance, taken in connection with evidence of mechanical pressure, especially if there be no history or evidence of syphilitic infection. It must be remembered, however, that sarcoma is not unlikely to occur in the young, a fact of great moment in connection with pressure-phenomena.

Under some circumstances the presence of an intrapleural effusion may aid in the recognition of pulmonary neoplasm; in others it may serve to conceal the lung-growth absolutely. It is held to be of aid in diagnosis if, after the evacuation of the fluid, the normal resonance does not return or dullness is recognized which extends above the level of the previous exudate, or if it has an irregular outline instead of the S-shaped curve seen in pleuritic effusion, or if, after aspiration, there be a dull area at the root of the lung while the remainder has become resonant. The foregoing considerations are all valuable but as a matter of fact their determination is by no means an easy matter at the bedside.

Wasting and a cancerous color of the skin cannot be relied upon since they do not occur so early or so markedly as in malignant disease of other viscera. When present they would, of course, possess much value.

In the third group of cases in which no suspicion of the growth within the lung is entertained or, if so, its presence is effectually hidden by some accidentally associated or secondary affection, there are no symptoms and no physical signs by which the tumor may be diagnosed. These are very apt to be instances of primary lung neoplasm or of a growth primary in a bronchus or bronchial lymph node, which is yet too small to make its presence known by pressure-effects. The real nature of the case is first revealed by the autopsy, or perchance is disclosed by the accidental detection of cancer cells in sputum or pleuritic effusion.

The inability to secure definite ætiological data for the obscure and perchance suddenly developing illness should render the diagnostician ever on the alert to detect some clew. A sudden hæmoptysis or a chronically bloody expectoration occurring in a person past middle age should arouse suspicion and should not be too readily attributed to tuberculosis. The more anomalous the general symptoms and local findings the more assiduously should the thought of malignant growth be kept in mind.

The question as to the particular variety of growth when one is believed present is still more difficult of answer. The nature of a coexisting neoplasm or of one that has been removed will, of course, furnish the clew. The age of the patient may also be of assistance, since sarcoma is more likely in the young and carcinoma in persons of middle age or beyond. Moreover the latter is more apt to undergo ulceration and yet, as far as this goes, we can only say that a growth that breaks down is not lymphosarcoma.

Differential diagnosis concerns mainly three affections, all of which have of necessity been touched on already. These are aneurysm of the thoracic aorta, tuberculosis of the lungs, and pleurisy with effusion of tuberculous or other but not malignant origin.

Aneurysm.—This occurs far more often in males than females. In the majority of cases there is history or some evidence of previous syphilis. There is apt to be a bruit or some modification of the vascular or heart sounds. Inequality of the pulses, localized pulsation, thrill, tracheal tug and displacement of the heart are all more likely to be present. Stridor especially is more likely than in primary lung tumor. Hæmoptysis is not so likely in aneurysm certainly in the early part of its course. Pressure-effects are apt to be greater than in a primary growth of the lungs, the mediastinum being wholly or comparatively uninvolved. Pressure-effects may change from time to time and sometimes depend largely upon posture. The X-ray is likely to show pulsation in the aneurysm.

Pulmonary Tuberculosis.—Pressure-effects are usually absent and dyspnœa is not marked. Tuberculosis is more probable in the young. General symptoms are apt, in early cases, to outrun the local signs and at a later stage are those of mixed infection. Pulmonary signs are likely to be more distinctly apical and posterior, often bilateral, while the râles are more clearly those of breaking-down lung tissue and cavity formation. The cachexia is different and the wasting is apt to show more plainly in the musculature and adipose tissue of the chest. Bacilli are likely to be present in the sputa. When both affections are present the malignant disease is very likely to escape recognition altogether.

Pleurisy with Effusion.—When independent of cancer the fluid is most likely to be serous. If it is hæmorrhagic it may belong to tuberculosis as well as to malignant growth. Removal of the fluid may be followed by reëxpansion of the lung and return of pulmonary resonance. Pressure-effects are likely to subside after aspiration and reëxpansion. In some cases there is evidence of tuberculosis in the apex of the same or opposite lung. For additional particulars see chapter on Pleurisy with Effusion.

Prognosis.—This is absolutely unfavorable. Carcinoma and the various forms of sarcoma lead eventually to death. One should be cautious, however, in his response to inquiries concerning the *duration* of the disease. Computing this from the date of recognizable symptoms it may be put down as from a few months to one or two years.

Treatment.—It goes without saying that we possess no specific means of cure for these unfortunate cases. Surgical operation is not safe and would scarcely be undertaken by the surgeon even were the physician to advise it. Treatments by means of the X-ray or radium offer no prospect of cure or even delay in the progress of the growth when this is so deeply situated. Accordingly, we are confined to such measures as will mitigate suffering and sustain powers of life. These will suggest themselves as occasion may arise.

SECTION III

DISEASES OF THE PLEURA

CHAPTER XXXV

ACUTE INFLAMMATION OF THE PLEURA

Pleuritis, or pleurisy as it is oftener called, is an exceedingly common affection and one whose onset is announced by such marked symptoms that it was recognized by physicians long before our present methods of diagnosis had come into vogue. Prior to the writings of Boerhave, however, in the seventeenth century pleurisy and pneumonia were frequently, nay quite generally, confounded. It was this great Dutch clinician who first attempted the clinical separation of these two diseases. Nevertheless, it was Laennec who succeeded in drawing so accurate a picture of the disease that subsequent clinicians have been able to add but little to his description.

Such has been the almost inconceivable advance in internal medicine during the past hundred years that to-day we are no longer concerned with questions pertaining to the clinical manifestations of pleurisy, its diagnosis and its prognosis, or, it might almost be added, to its treatment, but instead, with certain problems that have grown up very naturally out of the keen and vital interest taken in bacteriology. In other words, the questions that now chiefly concern us, with regard to pleuritis as well as other diseases of the respiratory organs, have to do with causation. For this reason we are interested also in its classification. From the standpoint of pathology the three varieties, dry pleurisy, sero-fibrinous pleurisy and empyema, possess a certain unity, being the manifestations of different degrees of inflammation; whereas in a bacteriological sense they may be, though are not always, distinct. An ideal classification would be in accordance with the ætiology of each case but for obvious reasons this is not yet possible.

We may, however, still divide pleurisies into primary and secondary, but we must abandon the old notion of an idiopathic pleuritis in a strict sense. How far an individual instance of this inflammation is primary or how far secondary, and in that sense a complication, cannot always be determined. The pleura may become inflamed in the course of an acute infectious disease without our always being able to determine whether the organism causing the general infection is also responsible for the local inflammation, or whether this latter be not the result of some added agency and hence a secondary process.

For the sake of convenience we must also recognize an acute and a chronic pleurisy although the difference is one of degree and not of kind, but it is not conducive to clearness to divide pleurisies according to their seat, nor, in my opinion, is it scientific to treat of the three forms as distinct entities because the character of the exudate differs. I have decided, therefore, to treat of all three forms in this one chapter. The old nomenclature cannot be wholly abandoned, nor can their clinical peculiarities be ignored, and hence such differences will be described under the terms applied to their exudative differences without losing sight of their pathological unity.

Ætiology. Predisposing Conditions.—No direct causative influence can be attributed to either *age* or *sex*, but children and young adults appear to be more often afflicted with pleural inflammations than are the old. Nevertheless, a low grade of pleuritis attended with sero-fibrinous or hæmorrhagic effusion is not infrequently met with in persons past middle age in association with and probably dependent upon certain affections seen in elderly people—chronic nephritis, hepatic cirrhosis and malignant disease. If, as is sometimes stated, males are more often affected than are females, the difference lies in the greater exposure of the former to the diseases to which pleurisy may be secondary.

Exposure to cold to which formerly so many cases of so-called idiopathic pleuritis were attributed is now believed to act as a determining factor by inducing nutritive or chemical disturbance within the pleural membrane, which favors the growth and development of bacteria brought to it in the lymph stream. As will be seen later on, so-called idiopathic, i. e., a primary pleurisy is in the majority of instances of tuberculous origin, and in some of such cases exposure merely serves to call forth the inflammation for which the membrane was prepared by the presence of miliary tubercles. In other instances in which the primary pleurisy is due to the pneumococcus the initial chill is probably mistaken for a chill of exposure.

Trauma.—Injury to the chest-wall is another predisposing condition. With regard to the influence of traumatism, however, we are forced to believe that, like exposure, it only prepares the soil for the growth of microorganisms by occasioning a nutritive alteration which lessens the resistance to bacteria on the part of the pleura. It may here be stated, also, that Netter believes that in 68 per cent of cases attributable to traumatism tuberculosis is the real cause of the pleurisy (Rosenbach).

Acute Infectious Diseases.—Herein is to be found the largest number of predisposing factors and at their head stand acute fibrinous pneumonia and tuberculosis. Every attack of pneumonia is attended with some degree of pleural inflammation from an unimportant fibrinous exudate to an empyema. Usually the pleuritis is on the same side as the pneumonia but not unfrequently the membrane covering the opposite lung is also affected. In pulmonary tuberculosis, also, the pleurisy may be dry and circumscribed or sero-fibrinous or purulent and involve the greater portion of the membrane.

Scarlatina, measles, diphtheria, typhoid fever, acute articular rheumatism, variola and even *gonorrhœa* (Jicinsky) may all be occasionally responsible for pleuritis. These will be referred to again in connection with the bacteriology of this particular affection. *Acute specific bronchitis* and, according to Rosenbach, *influenza* are rarely attended with inflammation of the pleura because they are usually confined to the bronchial mucosa. General infections, on the other hand, due to pyogenic bacteria are sometimes attended with purulent pleuritis.

Extension from Neighboring Structures.—The liability to involvement of the pleura from acute or chronic disease of adjacent parts has long been recognized as a predisposing cause of pleurisy. Perhaps the most frequent of these is pericarditis, the secondary inflammation of the pleura being either by direct extension or by way of the lymph channels. Localized purulent inflammations within the peritonæum may sometimes act in the same manner. The pus may rupture into the pleural cavity or burrow its way upward along the retroperitoneal tissues, or the germs may be carried directly to the pleural cavity through the chain of glands situated behind the peritonæum. Thus, I recall a case in a young man in whom the empyema which eventually burst through the bronchi was in all probability traceable to a peri-appendicular abscess that had formed some two months previously. Pulmonary abscess or gangrene, caries of a rib or vertebra, actinomycotic disease in the mediastinum or lung, cancer of the œsophagus, may all produce inflammation of the pleura, and in such cases the pleuritis is purulent or putrid.

Malignant growths in the lung or mediastinum frequently produce pleurisy, the resulting exudate being sero-fibrinous, hæmorrhagic or purulent. In such instances the pleura becomes involved by direct extension of the neoplasm. Cirrhosis of the liver may likewise be associated with pleuritis, but as this form of hepatic disease not seldom leads to tuberculosis it is very probable that the pleurisy is of this nature. In other instances it may be of the nature of a terminal infection as probably is the pleuritis seen in the last stages of chronic nephritis. I am inclined to think, also, that the pleural effusions met with in some cases of advanced cardiac disease and which are not always easily distinguished from hydrothorax, especially when they occur on the right side, are a manifestation of infection, since we know that terminal infections, e. g., terminal pneumonia, are not uncommon in cardiopaths. Gerhardt believes that such pleurisies are often of embolic origin.

Exciting Causes. *Bacteria.*—A great number of micro-organisms have been identified in pleuritic effusions, of which the chief are the tubercle bacillus, the diplococcus pneumoniæ and the streptococcus pyogenes. The first of these has received an immense amount of investigation because of the interest attaching to the ætiology of primary pleurisies. Under this head come the numerous cases of pleural inflammation with a sero-fibrinous exudate, which appear to arise spontaneously or idiopathically in persons seeming to enjoy robust health and hence have been ascribed to exposure or injury of the chest-wall.

This class of cases has been the object of special study in France, Germany, the United States and in fact everywhere. The fluid obtained by aspiration has been examined by every means known to bacteriologists: by microscopic examination, culture and inoculation experiments, and by clinical methods, among which is the investigation of subsequent history of persons known to have had spontaneous pleurisy. The results have been comparatively uniform, and warrant the conclusion that in the majority of instances the inflammation is of tuberculous origin. Thus 6 out of 15 animals, or 40 per cent, inoculated by Pansini with the exudate from an equal number of sero-fibrinous pleurisies became tuberculous, while Netter obtained the same percentage in 25 cases of pleurisy supposed due to exposure. Of 16 cases in persons known to be tuberculous 8, or 50 per cent, proved of this origin. Aschoff made inoculation experiments in 37 cases which microscopically and culturally had proven negative and demonstrated their tuberculous origin in 68 per cent. Eichhorst who used at least 15 c.c. for his inoculations obtained positive results in more than 62 per cent. By the employment of still larger amounts, sometimes as much as 300 c.c. being injected into a single animal, Le Damany demonstrated the tuberculous nature of 47 out of 55 cases of primary pleurisies or 85.4 per cent.

In a clinical study of 90 cases of pleurisy observed by his father between 1849 and 1879 Henry I. Bowditch, Jr., found that 32 or 35 per cent ultimately died of tuberculosis, while of 131 pleurisies of all types examined *post mortem* Osler found that 32 or about 25 per cent were of tuberculous origin. French observers, Landouzy, Germain Sée, Strauss and others hold to the tuberculous origin of these cases of so-called idiopathic pleurisy, some going so far as to declare that 70 to 75 per cent of them are of this nature. Fowler believes that if the pleura lining the interlobar fissures were carefully examined for tubercles *post mortem* the great majority of the cases would show the presence of miliary tubercles. From the evidence, therefore, we must abandon the notion of an idiopathic pleuritis.

The pneumococcus of Fraenkel is another cause of some cases of primary inflammation of the pleura, the exudate being commonly though not invariably purulent. These instances are met with most often in children but may occur in adults. In the majority of cases, however, of pneumococcus pleuritis the inflammation is secondary. This form of empyema is usually benign.

The streptococcus has been demonstrated in the secondary pleurisies of a great variety of disorders—pulmonary abscess, broncho-pneumonia, traumatic and other inflammations of the chest-wall and neck, scarlatina, and occasionally in association with measles, diphtheria and pertussis. In scarlet fever and diphtheria, however, the streptococcus is frequently associated with other organisms (Councilman, Mallory, Pearce).

Among the other bacteria that occasionally cause pleuritis either alone or in combination are the staphylococcus pyogenes aureus, the colon bacillus, Friedländer's pneumobacillus, the diphtheria bacillus, micrococcus tetragenus, and typhoid bacillus (Charrin, Rogers, Sahli, Frenet). Pure cultures of the typhoid bacillus were obtained in two cases by Achard. Jicinsky has reported two cases of gonococcus infection in which a pleurisy occurred, the exudate of which contained diplococci believed to be gonococci.

In conclusion, I may say that in the light of the above and many other investigations I have no doubt of the bacterial origin of all cases of pleuritis, of whatever nature the exudate. Whenever I find a case of pleurisy giving no history of a predisposing factor I set it down to either the tubercle bacillus or the pneumococcus, and if it be a sero-fibrinous one I regard it as due to the former until proven otherwise.

Morbid Anatomy.—The earliest inflammatory change in the pleura, as well as in other serous membranes, is an opacity and roughening in consequence of which the membrane loses its luster in certain areas. At the same time the vessels of the subserous layer are injected. The endothelial cells speedily undergo coagulation necrosis, which, together with the pouring out of fibrin from the vessels, forms the exudate (Fig. 97). This at first contains very little if any serum but being plastic is forced by the rubbing together of the two surfaces of the pleuræ to assume a bandlike or reticular arrangement, between the ribbons of which and beneath which the membrane appears ecchymotic. The costal layer is the one chiefly concerned in this inflammatory process.

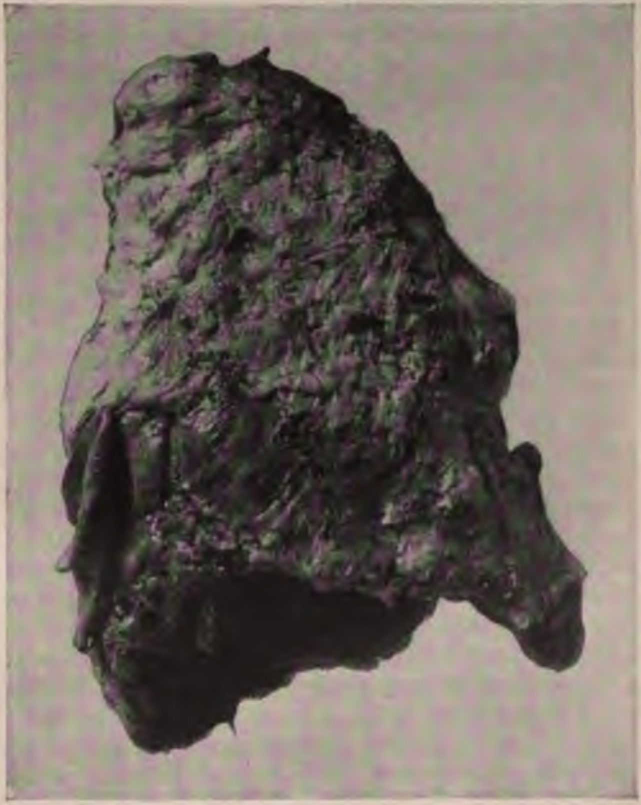


Fig. 97.—External surface of lung with acute fibrinous pleuritis.

Adhesions now form between the visceral and costal layers of the pleura at those parts, as the apex and between the lung and diaphragm, where the respiratory movements are limited, whereas along the margins there are no adhesions or the fibrin is drawn out into loose bands or ribbons. If the exudate is abundant the surface of the pleura may be covered by a more or less thick uniform layer. In tuberculosis these changes are most marked over the upper lobe in consequence, probably, of the limited excursion movements of the apex as compared with the base.

As the activity of the process abates either one of two changes may occur. The exudate may undergo fatty degeneration and become absorbed by the rich network of vessels newly formed and existing in the succulent subserous tissues of the costal layer, or the fluid portion of the exudate may be absorbed leaving the fibrin and cellular elements behind. In the former event the pleura returns to very nearly its normal condition and the only evidence of what was an intense inflammatory process is a slight opacity or thickening of the membrane. If the exudate does not undergo absorption, the pleural cavity may be totally or partially obliterated, the latter being far the more frequent. The two pleural layers in certain regions are united by a firm, more or less thick stratum of fibrin, or they are bound by a loose connective tissue which, with the lapse of time, becomes vascularized and organized. The process just described is known as *dry, plastic* or *fibrinous pleurisy.*

Sero-fibrinous Pleurisy or **Pleurisy with Effusion.**—In this form the initial changes are identical with those just depicted, but in addition there is a more or less abundant effusion of the serum together with the products of inflammation. The exudate differs in appearance and chemical characters according to the degree of irritation and the resulting preponderance of its constituents. It may be relatively thin, clear and opalescent containing very little fibrin, albumin and cellular elements, or viscid and turbid or hæmorrhagic, depending upon the number and proportion of the cells present. The effusion is especially apt to be bloody in pleurisies of cancerous or tuberculous origin, in chronic alcoholism, in scurvy and in dyscrasias of other cause.

The specific gravity of a serous exudate in the pleural cavity may vary within rather wide limits. Fowler gives it as between 1.005 and 1.030, while Rosenbach puts the maximum at 1.035. J. L. Miller in a very helpful contribution to this subject has tabulated the figures given by several investigators to which he has added a series of his own. I avail myself, therefore, of the facts and figures therein contained. The differences between pleural exudates and transudates will be found in the chapter dealing with hydrothorax.

Of 22 cases of sero-fibrinous pleuritis investigated by Neuenkirchen the range of specific gravity was from 1.014 to 1.022; Ott found it to vary between 1.009 and 1.026; Miller in 27 cases obtained figures ranging between 1.014 and 1.025. In a case recently observed by myself and narrated in the chapter mentioned above I found 1.028 to be the specific gravity of the fluid obtained from the right pleural sac; from the left it was 1.022. This deviation agrees with Miller's statement that the exudate in the right pleural cavity may be somewhat heavier than that in the left.

Tuberculous and cancerous pleurisies are said to show the highest specific gravity with figures in favor of the former. It would seem, therefore, that the maximum figures given by Fowler and Rosenbach may not be too high; the minimum of 1.005 is, however, too low in my opinion, and so well within the limits of the figures given for transudates that such a specific gravity might justly be considered to place a fluid in the category of a transudate and not an exudate.

The *percentage of albumin* in a pleuritic exudate also varies and depends upon a number of factors. It is determined largely by the permeability of the capillary walls and by the state of the patient's blood as regards this constituent. In 26 cases investigated by Runeberg the amount of albumin ranged between 6.04 per cent and 4.78 per cent; while Ott found a maximum of 8.9 per cent and a minimum of 2.4 per cent. In the differential diagnosis of hydrothorax will be found Reuss's formula for the estimation of albumin, which has been tested by various investigators and found to be a simple and accurate method of determining the percentage of albumin from the specific gravity.

The *fibrin percentage* is also very variable, being greatest in pneumococcus pleurisies. A sero-fibrinous effusion of this nature may contain abundant flocculi, while a tuberculous exudate is apt to be poor in fibrin.

The *color* of a serous exudate is generally that of blood serum, but it may have a peculiar yellowish hue from admixture with bile in jaundice; and Guttmann and Gerhardt are said to have found it bluish from the presence of indican.

The *amount* of a pleuritic effusion varies enormously in different cases from a few ounces to many quarts, as many as 280 fluid ounces having been withdrawn in a single instance (Fowler). Rosenbach gives the average as from 1 to 2.5 liters.

Physical exploration of the chest during the formative stage of a serous effusion shows that it collects first in the lower portion of the pleural cavity and thence creeps gradually upward. This is not as might be expected wholly in consequence of gravitation; but, as explained by Rosenbach, fluid assembles first at the bottom because the resistance presented by the lung is less. As the exudate increases in amount it makes room for itself by displacing the neighboring organs. The mediastinum is the first to show the effect of this pressure and hence the apex-beat becomes pushed farther and farther toward the opposite side.

The resistance offered by the lung under normal circumstances is considerable, but as the exudate advances upward the organ undergoes corresponding collapse, until, in massive effusions, the lung may become completely atelectatic and may be reduced to the size of a man's fist (Fig. 44). In such instances the lung occupies a small space at the upper and back part of the chest cavity, being suspended by its root as an inert mass. With the lapse of time it may become fibrous and present a brownish appearance, yet, despite its shrunken size, show the interlobar markings.

If the pleural membranes be examined *post mortem* there is generally found a deposit of more or less fibrin upon the surfaces of the two membranes and these may, especially in pneumococcus pleuritis, be united by easily broken-down adhesions along the margins of the exudate. In tuberculous pleurisies of this kind there may or may not be discovered miliary nodules on the visceral pleura. Fowler states that such tubercles are especially likely to be present in the interlobar fissures.

Purulent Pleurisy; Empyema.—In this variety of pleuritis the exudate is rich in cellular elements and hence is thick and turbid and contains a variable proportion of fibrin. In pneumococcus empyema the exudate is remarkably rich in this latter constituent and tends to sacculation; hence this form of purulent pleurisy is nearly always encysted. The amount of the exudate is not usually large and in color it may vary from a light yellow to a greenish-yellow. On the other hand, a streptococcus empyema is thinner, rarely encysted, of a light yellow hue, and may be very abundant. The formation of pus indicates either a more intense virulence on the part of the germs or less resistance on the part of the host. The exudate may be so rich in cells as to be purulent from the beginning or, at first serous, it may speedily become purulent. Micro-organisms are usually discoverable in the exudate along with the products of inflammation.

Putrid Pleurisy.—A purulent exudate is generally free from odor, but in some cases when secondary to gangrene of the lung or to purulent inflammations within the abdomen, either by extension or perforation, it may possess a cadaveric or fæcal odor. In such instances it is known as putrid pleurisy. The exudate is usually thin and of a greenish-yellow, brownish-yellow or chocolate color. Plugs made up of detritus and micro-organisms may be seen with the naked eye and microscopically are found disintegrated cells, crystals of hæmatin, needles of fatty acids, free fat, leucin, tyrosin, cholesterin and micro-organisms. Rosenbach is authority for the statement that a sero-fibrinous effusion may occasionally possess a putrescent odor.

Hæmorrhagic pleurisy is one in which, owing to abnormal permeability of the vessel walls, the red blood cells are permitted to escape by diapedesis into the exudate. This may result from unusual violence of the inflammatory process or it may denote a local or a general tissue weakness. The color of the exudate in these cases may vary from a slight tingeing with red to the hue of dark venous blood as in endothelial cancer of the pleura (Fraenkel). The conditions favoring a hæmorrhagic pleurisy have been already stated.

Multilocular pleurisy is one in which the deposit of fibrin upon the surface of the pleura divides the sac into a series of pockets containing exudate. Although in most instances the contents of these encysted cavities are uniform, still it may happen that in one there is serum while in another there is pus. The spaces thus formed may be wholly separate or they may communicate one with another by means of tortuous passages.

Just as a pure or nearly pure fibrinous exudate may undergo absorption under certain conditions, so a sero-fibrinous or purulent one may become absorbed or it may persist and the fibrinous portion be deposited in thick plates or bands. An empyema may eventually become transformed by fatty degeneration of its cellular elements into a cheesy material and lime salts being deposited therein, it may be converted into calcareous plates. Absorption is hindered by the early formation on the two pleural surfaces of dense layers of exudate in consequence of the obstruction thus furnished to the circulation in the lymph channels to and from the pleuræ. The more violent the inflammation also the more serious the changes in the membrane and the less likely is spontaneous removal of the exudate.

The **direct results** of a pleuritic effusion are retraction of the lung and displacement of adjacent viscera, heart, liver, etc. The retraction of the lung is not the result of compression but is the expression of the natural tendency to contraction inherent in the pulmonary parenchyma. In massive effusions, therefore, the lung may be found completely collapsed (Fig. 44). and, in cases of long standing, airless and fibroid. Furthermore, the envelopment of the contracted lung by dense, often-organized fibrin prevents its reëxpansion after artificial removal of the exudate.

If a purulent exudate be not removed either by absorption or by the surgeon, it is apt to lead to necrotic processes and, if such necrosis affects the pulmonary pleura, rupture takes place with escape of the pus through the bronchi or, very rarely, through the diaphragm. If the necrosis be in the costal pleura the exudate may make its way through the intercostal tissues and appear beneath the muscles or integument as a fluctuating tumor, or it may burst through the skin and appear externally, the so-called *empyema necessitatis.*

Furthermore, the prolonged retention of a putrid or merely purulent exudate may lead to septicæmia through absorption of toxins, or to an actual pyæmia. In cases of intense suppurative inflammation a so-called pyogenic membrane may be formed on the pleural surfaces, in consequence of which, pus continues to be secreted each time the exudate is removed. In such cases death is very likely to take place from exhaustion or after a long time from amyloid disease of liver, kidneys and other organs.

The Mechanical Effects of Intrapleural Exudates.—As already stated the accumulation of fluid within the pleural sac is followed by retraction and eventual atelectasis, either of the lower portion of the organ in moderate effusions or of the entire viscus in massive collections. This collapsed state is not the result of compression but is, as generally expressed, the exaggerated manifestation of the elasticity of the lung. In other words, it is an exaggeration of its tendency to expiratory retraction.

Rosenbach argues that the collapse of the lung under these conditions has nothing to do with its elasticity, but results from the fact that the presence of the exudate hinders perfect contraction of the inspiratory muscles and hence reënforces the pulmonary tonus by virtue of which the lung is in a state of retractile tension. Inspiration is not caused by traction on the lung outward, i. e., extension, but by internal air-pressure (the pulmonary tonus or tendency to concentric retraction being relaxed at the proper moment, namely, at the commencement of the inspiratory act) together with increase in the capacity of the thorax produced by contraction of the inspiratory muscles. If now for any reason the antagonism between the tonus or pull of the inspiratory muscles and the tendency to concentric retraction or tonus on the part of the lung is interfered with, as by a foreign body in the pleural cavity, then relaxation of the lung's inherent tendency to concentric retraction does not occur as during inspiration and the lung collapses.

Furthermore, this atelectasis is intensified by absorption of the residual air in consequence of the abolition of the interchange of gases, as we are wont to say, incident to proper respiratory movements. The organ therefore becomes a compact airless mass of a slaty-gray color occupying the upper and posterior portion of the thoracic cavity. In response to the law of the development of fibrous tissue in a disused organ the lung in time becomes fibroid and unable to reëxpand after removal of the exudate, even if not bound down by a firm layer of fibrin.

Displacement of Adjacent Organs.—A necessary consequence of the accumulation of fluid or air in the pleural cavity is the dislocation of neighboring viscera, chiefly liver and heart. This was formerly attributed to direct pressure, but according to modern views this is erroneous. The liver may be displaced downward *in toto*, the organ maintaining its normal relation to its planes of axis or it may undergo a rotation on its transverse axis, that is, about its suspensory ligament, in such a manner that the right lobe is depressed while the left lobe is elevated.

Rosenbach holds that depression of the liver cannot be due to direct pressure on the diaphragm and flattening of this muscle, since that would necessarily abolish its respiratory play. Instead, there is an augmentation of work on the part of the sound lung and this, he argues, causes a flattening of the great inspiratory muscle, the diaphragm, which probably accounts for the rotation of the liver together with its downward displacement.

Displacement of the Heart.—Unless this organ be fixed by adhesions it is always displaced more or less toward the side opposite to that occupied by a pleuritic exudate. The heart is moved over *in toto* but naturally to a greater extent at its apex than at its base where it is held by the large vessels. Consequently the apex is tilted somewhat upward, though without much if any rotation of the organ on its longitudinal axis. There is also kinking of the great vessels (Rosenbach). As in the case of the liver, this dislocation of the heart is not the result of pressure as such, but is due to traction exerted by the sound lung in consequence of the increased stimulus to contraction felt by the lung.

Widening of the thorax is another effect of pleuritic effusions, the degree of dilatation of the thoracic cavity being proportionate as a rule to the amount of the exudate. The elasticity of the parietes also has much to do with the distention of the chest and hence this is more apparent in the young. In persons whose costal cartilages have become rigid from deposit of lime salts the increase in the diameters of the affected half of the thorax may be slight. If the inflammatory process be very intense, as in cases of long standing, the intercostal muscles become paretic and hence the interspaces on the diseased side may bulge perceptibly. Indeed, fluctuation may sometimes be elicited over the widened and prominent intercostal spaces.

Remote Effects.—These are seen in the circulation, the respiration and in visceral function. As will be stated in considering the pulse, there is a relative arterial anæmia shown by the diminution in volume of the pulse and decrease of blood-pressure. Several factors probably are responsible for this. The interference with normal respiration incident to collapse of one lung would naturally lessen the aspiratory effect of full inspiration on the flow of blood out of the superficial into the great intrathoracic veins, while the right auricle, hindered in its diastolic relaxation by the altered conditions resulting from cardiac displacement as well as in consequence of lessened supply from the great veins, receives less blood than normal. The right ventricle is thereby deprived of some of the blood it should normally receive. But in addition it is subjected to strain in consequence of the atelectasis of the lung and undergoes more or less dilatation.

This condition is also favored by the diminished supply of nutriment and of oxygen to the myocardium. Consequently, the flow through the lungs into the left heart is impeded. The organ is stimulated to increased frequency of contraction, and a lessened quantity of blood is discharged with each systole into the aortic system. This state of things reacts upon the venous circulation, and stasis results in the portal and other abdominal vessels, this stasis being furthered by the diminution of the pumping action of normal diaphragmatic play. We have, therefore an abnormally frequent, small and irritable pulse, this latter quality being shown by increased acceleration of the heart-beats whenever the patient makes physical exertion.

The respirations become more or less accelerated and despite their increased frequency hæmatosis is diminished. Dyspnœa is the necessary result, which embarrassment of respiration, even when the individual is at rest, may amount to actual orthopnœa.

The resultant effect of these conditions on the functions of the various organs becomes apparent. The processes of digestion and assimilation are interfered with and secretions and excretions are lessened. In particular is the excretory function of the kidneys affected so that the urine is lessened in amount and concentrated. The entire organism suffers therefore, in consequence of the circulatory and respiratory disturbance, to say nothing about the injurious effects of the inflammatory process itself.

Symptoms.—The onset of acute pleuritis may be insidious or abrupt. In the former event the individual who may have thought himself well, or who may have been running down in health, notices *malaise*, anorexia and perhaps headache which he attributes to some digestive disorder or "biliousness" but still keeps at work. His feeling of weakness continues or increases and at length he seeks medical advice. The physician finds the temperature more or less raised and, if he examines the chest, discovers friction-sounds or dullness at the lower back part of one lung and makes a diagnosis of pleurisy.

Both he and his patient are surprised by the finding and wonder what may have caused the inflammation. In some cases no ætiological factor can be elicited while in others the patient at length recalls that near the commencement of his indisposition he felt chilly or was exposed to a draught, whereupon the physician promptly sets it down as due to exposure. It is precisely this class of cases that renders me suspicious of a tuberculous origin.

In another set of cases the pleuritis, which is also primary, begins more or less abruptly either in the midst of apparently good health or after a period of failing strength and disinclination to the performance of his usual duties.

The *initial symptom* in these instances is a *chill* or a succession of chilly or creepy sensations up and down the back. This is very apt to be accompanied or speedily followed by a sharp *pain or stitch in the side* which may cause the individual to catch his breath and cry out. Or the pain may partake more of the character of a dull ache or feeling of soreness.

The **pain** is most commonly felt below one or the other nipple, being in such instances quite circumscribed. It may, however, radiate around the lateral aspect of the chest to the back or may be reflected along the nerves distributed to the abdomen. It is not transient in duration but persists or grows in intensity and may become unbearable. The pain is aggravated by inspiration and especially by the act of coughing so that the patient endeavors to breathe as superficially as possible and in particular to suppress his cough. This symptom usually abates in severity after the formation of the exudate but in some cases remains severe even after a considerable sero-fibrinous effusion has collected. This circumstance makes me doubt its being due to rubbing together of the two inflamed pleural surfaces. It is a neuralgia of the intercostal nerves.

Cough may or may not be an early symptom. Indeed there is much diversity in the occurrence and severity of this feature. Unquestionably cough may be entirely absent in some cases, which corroborates the view that irritation of the pleura itself does not excite the reflex phenomena known as cough. When present it is probably due to an associated bronchitis or to a collection of mucus in the air-passages. It is apt to be called forth by an attempt at deep inspiration, especially after pulmonary collapse from a copious effusion.

Expectoration is not generally present in the inflammatory stage or, if so, is scanty and catarrhal.

Fever is an early symptom in most cases but may be wholly absent (Rosenbach). I have seen cases of pleurisy in persons with cardio-vascular or renal disease who did not manifest rise of temperature and in such the pleural inflammation sets in so mildly as hardly to merit the term of acute; but I cannot now recall an instance of primary acute pleuritis in young persons in which more or less pyrexia was not present.

The range of temperature may be slight and irregular, or the fever may be of the continued type with no marked fluctuations. Moreover, the degree of pyrexia and even its character does not always seem to depend upon the character of the exudate, for, on the one hand a fibrinous exudate may be attended with high fever and on the other, an empyema is not always manifested by a high or irregular temperature curve. This feature appears to be determined by the intensity of the local reaction, that is, by the virulence of the causative agent, plus the vigor of reaction on the part of the tissues.

Such are the leading features of an acute pleuritis, particularly of the dry or plastic variety. In addition there are certain local symptoms or signs which will be detailed under the head of physical signs. These are more or less restriction in the respiratory movement of the affected lung, normal or impaired pulmonary resonance over some portion, usually the base of one lung, and friction râles audible during inspirium or also expirium.

Other subordinate symptoms of constitutional disturbance are present in varying proportion as determined by the intensity and duration of the process and the general strength or vigor of the individual. If the pleurisy be quite circumscribed, as at an apex, e. g., in cases of tuberculosis, the symptoms just described may be mild and the health of the patient may not appear seriously affected. The symptoms subside in a few days and convalescence proceeds satisfactorily.

At other times the local and constitutional symptoms continue for two or three weeks and the individual loses strength and color. Appetite remains poor, constipation results, the urine is scanty and perhaps albuminous, the pulse is rapid and small, respirations are decidedly dyspnœic, largely because of pain, and the individual grows nervous and irritable. In such cases, however, one is doubtful of the process being merely fibrinous. The impairment of resonance is too pronounced although not intense and as a matter of fact, exploratory puncture at this time is likely to reveal serum in small quantity. The case is one of acute pleurisy in which the exudate is rather too copious to be strictly a plastic pleuritis and yet not large enough to bring it clearly under the head of the variety soon to be considered.

Dry pleurisies, strictly speaking, are met with most often in the course of chronic pulmonary tuberculosis when the symptoms they produce are usually slight and transient. They may also occur in connection with acute pneumonia, in which event the symptoms referable to the pleuritis are obscured by, or regarded as, a legitimate part of the clinical picture of the pneumonia.

The **condition of the blood** in acute pleuritis is highly interesting and important from the standpoint of differential diagnosis. Morse's investigations showed that in primary pleurisy the leucocytes are not much increased. Of 224 counts made by him they exceeded 10,000 in only 13 instances, 9 of these being in a single case due to pneumococcus infection. Morse concluded that the number of the leucocytes bears no relation to the degree of fever, to the character of the exudate unless this be distinctly purulent, or to the amount of the fluid.

For my part, I should regard an absence of increased leucocytosis as an indication of the tuberculous nature of the case. Whereas should a serous exudate be associated with marked increase in the leucocyte count I should regard it as probably one of pneumococcus origin or as indicating some complication. *A priori*, one would expect an empyema to produce marked leucocytosis.

The *urine* possesses the characters usual in febrile affections. It is scanty, of high specific gravity and may or may not contain albumin. In the strictly inflammatory stage albumin and casts probably indicate a symptomatic nephritis produced by the elimination of bacteria or toxic substances. When, however, an abundant exudate has led to pressure-effects the albuminuria is probably the result of stasis in the renal vessels.

Sero-fibrinous Pleuritis.—The onset and early symptoms are essentially the same as in the strictly plastic variety. In tuberculous cases, particularly, the disease may creep in so insidiously as to escape recognition until after the formation of considerable exudate. Such was the case in a young physician who, although not feeling quite up to par, was yet attending to his ordinary duties. On examination of the chest I found an easily recognizable serous effusion in the left pleural cavity reaching to the level of the fifth costal cartilage.

The symptoms of active inflammation may speedily become subordinate to those of mechanical pressure already described, or pain, cough and fever may dominate the scene. In some such instances the physician may be surprised at the amount of fluid indicated by physical examination of the chest. *Dyspnœa* is now apt to be a marked feature and is shown by frequent and laborious respirations. The sufferer lies more or less elevated and on the back or is partially turned on the affected side, since this position eases pain and permits free play of the opposite lung. In massive effusions there may be orthopnœa.

In some cases in which the exudate is not sufficient to interfere materially with breathing, yet in which the respirations are greatly accelerated, it is evident that the pain is the chief cause of the dyspnœa. I am satisfied that nervous apprehension on the part of the patient may increase the frequency of respiration.

Thus I recall a young woman who had an extensive apparently fibrinous pleurisy of the right side. Pressure-effects were not apparent and yet her difficulty of breathing was distressing. She was given a hypodermic of ¼ morphine and almost directly went into so profound a sleep that I became alarmed and resorted to measures to counteract the narcosis.

In the cases in which respiratory embarrassment appears due largely to the pressure-effects the dyspnœa is immediately intensified by the slightest exertion. In other instances remarkable tolerance of the presence of a serous effusion is manifested. This is very apt to be the case when the fluid accumulates slowly and when in addition pain is not a marked symptom. Probably also the vigor of the individual has some influence in creating this tolerance of pressure.

The *pulse* is usually accelerated and of reduced volume, the artery being appreciably contracted. In the stage of active inflammation the pulse may be tense but when, in consequence of intrapleural pressure, the amount of blood expelled by the left ventricle is reduced, arterial blood-pressure falls and the pulse becomes correspondingly feeble. Its rhythm may be regular but is apt to be disturbed by physical exertion and it may be intermittent. This is particularly so when, after a considerable duration of the exudate, the myocardium suffers in its integrity.

The patient suffering from acute sero-fibrinous pleurisy may look pale and anxious or the countenance may be flushed and the vessels be injected. This flushing of the face may be the result of high fever or of interference with the emptying of the veins of the head and neck. In such instances there may be headache. Sleep is also apt to be disturbed.

In this early stage of sero-fibrinous pleurisy there may be considerable sweating which, according to Rosenbach, may be paroxysmal. I observed a truly colliquative sweating recently in an old man who developed a right side pleuritic effusion after several months of cardiac inadequacy associated with vascular and renal changes. He had no pyrexia but perspired in a most uncomfortable manner so that we were obliged to give him atropine.

In such a case this symptom may be due to loss of tone on the part of the vasomotor nerves supplying the skin; for in the absence of pyrexia one would hardly attribute sweating to a compensatory attempt at elimination of toxins through the skin.

The loss of appetite, the constipation, the scanty urine, the loss of strength are all remote effects hardly separable from the presence of a serous or serofibrinous exudate in the pleural cavity.

If now the thorax and upper abdominal zone be examined they are seen to be increased in diameter on one side, the degree of the prominence depending upon the bulk and duration of the exudate. This appearance is particularly marked in children. Not infrequently, moreover, the patient complains of pain in the epigastric or hepatic region and palpation of the liver may call forth an expression of soreness or tenderness in consequence of congestion of the viscus.

Examination discovers signs of fluid distention of the chest. The position of the apex and the results of percussion denote displacement of the heart toward the sound side, and the lung may furnish evidence of more or less compensatory emphysema and increased functional activity.

A number of years ago I encountered a series of cases which illustrated the features and some of the problems of certain primary pleurisies. They may with profit, I think, be briefly narrated. Three were instances of tuberculous pleuritis while the fourth was of doubtful ætiology.

A young man of about twenty was brought to me by his physician for diagnosis. The history was meager, being, in effect, that a few days before he consulted the doctor on account of pain in the right side of the chest and fever. On examination physical signs were found which proved that the illness was of pulmonary origin, but of just what nature did not seem quite clear.

The young man declared that he did not feel sick, and yet his temperature was ascertained to be 101° F. The pulse was accelerated but the respirations were not at all difficult, being only somewhat shallow. On examination of the chest the right lung was markedly dull from the third rib down to the base in front and from a corresponding point to the base behind. The note was not uniformly dull throughout, but increased in this respect from above downward so that toward the base it became absolutely flat. The portion of the lung above was not tympanitic but impaired. The line of dullness did not shift with change of posture.

Over the upper portion of this dull area were moderately coarse crackling râles of a dry quality, most distinct on inspiration and unaffected by cough. Below, the breath-sounds were enfeebled and of a somewhat bronchial character. On the back râles were indistinct, and toward the base breathing was scarcely audible. At the apex the respiratory murmur was harsh. Pectoral fremitus over the area of dullness was feeble or absent, corresponding to the character of the percussion-note. The rather feeble apex-beat was at the nipple line and the liver was not palpable.

The opinion was given that this was a case of pleurisy with effusion and ought to be tapped. For verification of the diagnosis an exploratory puncture was made then and there, with the result of obtaining a hypodermic syringeful of clear serum. Arrangements were then made for an aspiration of the fluid next day. Accordingly, the operation was performed in my presence. To my surprise only 20 ounces could be obtained; and afterwards the physical signs remained practically the same as before.

The patient was kept in bed in spite of his protest, he declaring all the time that he felt well enough to get up and that he wanted to go out to play baseball. It was hoped that the exudate might become absorbed, but when, at the end of another week, the general and local condition remained *in statu quo* another aspiration was decided upon. The temperature all this time was running not far from 101° F. and very little cough or anything else was complained of by the patient. The second tapping resulted as did the first, only about 20 ounces being withdrawn and local signs being unchanged. In particular the position of the apex-beat did not move in toward the sternum.

I did not see this patient again for a year or more, but learned from the attending physician that in the course of several weeks health was so far restored as to warrant the resumption of his ordinary mode of life. When at length the young man again consulted me I discovered that he had a cough with scanty purulent sputum containing tubercle bacilli, had lost in weight, was running a moderate pyrexia and showed slight dullness with subcrepitant râles and broncho-vesicular breathing at the left apex. There was still some loss of resonance over the right lung, but this was not so marked as a year or so previously. He was sent to Asheville, N. C., where he ultimately died of pulmonary tuberculosis.

In this instance the degree of dullness led me to assume the presence of far more fluid than was obtained at either aspiration. Only three explanations are apparent. Either there was more than one pocket of fluid or there was consolidation together with effusion, or the exudate was exceedingly rich in fibrine, unusually so for a tuberculous pleurisy. I now, as then, incline to the view that a pneumonic infiltration existed together with the pleurisy. Else why did the lung clear up in part and why did it not show more decided retraction than was noticed at the time his opposite apex was found diseased?

The fact of the discordance between the only slight displacement outward and the extensive dullness on the right should have led me to assume the presence of but a small amount of effusion and should have saved me the very unpleasant surprise experienced at the time of the first tapping.

The second case was that of a lad of twelve years whom I saw at Evanston, Illinois, in consultation with the family physician, Dr. E. H. Webster. The boy's maternal uncle lay ill in the house at the time with pulmonary tuberculosis. The little patient had been ill a week or more, having come down rather suddenly though with what initial symptoms I do not now recall, the particulars being given from memory. He was in bed, cough not marked, temperature about 102° F., no special pain, no noticeable dyspnœa though breathing was accelerated.

The right lung presented signs very similar to those in the case just narrated; dullness from the upper third to the base and more intense below than above; diminished fremitus, friction râles just above the upper line of dullness and feeble, somewhat bronchial, respiratory sounds at base in front and behind; no very apparent displacement of the heart outward and no dislocation of the liver as determinable by palpation.

Exploratory puncture sustained the diagnosis of pleurisy with serofibrinous effusion which latter, however, was not believed to be large, and, if I am not mistaken, Dr. Webster did not succeed in withdrawing more than 10 to 12 ounces. The local findings did not change after the aspiration and the case dragged on for a number of weeks longer.

Improvement was slow but at length the boy removed to a ranch in southern Kansas where he stayed for a year or more and is said to have regained full health. I have not seen him again but understand that the lung did not entirely clear up, the same as in the former case.

Mrs. F., age twenty-five, was referred to me by Dr. H. T. Byford January 9, 1901, having been operated upon for tuberculosis of the ovaries and ascites in the November preceding. Since the operation her weight had increased 10 pounds, being 110 pounds at the time of my examination. There was nothing of special note in her appearance except that she was thin and rather pale. Family history of tuberculosis was denied, her chief complaint was flatulent indigestion, although she admitted being not very strong.

Examination of the chest revealed a not very resonant yet not distinctly impaired percussion-note at either apex, particularly the left, and here inspiration was harsh and jerky. Her temperature was not elevated, the pulse not unduly accelerated, and she had no cough. Nevertheless latent tuberculosis of the left apex was suspected, and the patient was ordered to drink a quart of milk daily, to take six raw eggs in addition to her three ordinary meals, to get as much fresh air as was possible under the circumstances, and to take a morning bath of cold water followed by a brisk rub. Upon the increased nourishment she improved rapidly, gaining 10 pounds in weight by February 27th, at which time all medicines were stopped. She now reported that she had never felt so well in her life and was digesting her food perfectly.

I did not see her for three weeks when, on March 18th, she appeared in my office with the following history: on Monday, March 11th, she felt as well as usual, but on the next day, Tuesday, she felt chilly, had sore, aching pains in the right side of chest but no cough. On Thursday she noticed shortness of breath with sudden catching pains in right side but very little cough. On Monday, March 18th, at my office, her temperature was 100.4° F. in the mouth, respirations 20, and pulse 115.

Examination showed the heart's impulse diffused and reaching about two inches outside the nipple line, the right half of the thorax very slightly fuller than the left with defective respiratory movements. To the third rib in front and in the suprascapular region the percussion-note was dull, while below there was flatness reaching to the extreme limits of that half of the chest. Pectoral fremitus was not abolished but was rather less pronounced over the base, and the voice-sounds were somewhat less intense, but everywhere heard distinctly. The breath-sounds were bronchial, plainly audible above the third interspace in front, and feebly audible below and over the back; there were no râles. Liver dullness extended below the costal arch and the organ could be indistinctly palpated.

The patient was sent home and ordered to remain in bed, and she was given $\frac{1}{30}$ gr. strychnine four times a day. On Sunday, March 24th, her condition remained practically the same but she complained of inability to sleep because, whenever she fell asleep she was soon wakened by a sense of smothering. The position of the heart seemed to be the same as a week before, and the entire right half of the thorax was flat below the level of the third rib in front, while above this point dullness seemed to have increased somewhat. Dullness passed across the median line in front to the left sternal border.

Pleuritic effusion was unmistakably present in the right side producing signs of pressure on the heart and liver, but the question also arose, Was there not, in addition, pneumonic consolidation? The intense dullness from apex to base and from left sternal border to spinal column seemed to indicate that the right pleural cavity was entirely filled with fluid. The side did not bulge, however, nor were the intercostal spaces so smoothed out as would have been expected in case of a massive effusion. Pectoral fremitus was everywhere feebly present and bronchial breath-sounds were faintly audible over the back.

It seemed reasonable, therefore, to assume a consolidation of the lung in addition to pleuritic effusion and not merely pulmonary collapse. Notwithstanding the nocturnal dyspnœa there did not appear to be sufficient embarrassment of circulation and respiration to render aspiration necessary at that time. This was performed a few days later, however, and to my surprise only 16 ounces of clear serum were obtained. As in the other cases the physical signs persisted very nearly or quite as before. Her breathing was relieved.

The patient was turned over to the care of a neighborhood physician and I did not see her again for many months. From this practitioner I learned that before convalescence was fully established a second attack of pleurisy, but this time on the left side, took place. Aspiration again resulted in the withdrawal of only 12 ounces of sero-fibrinous fluid. Recovery of health ultimately took place.

When I again examined this woman both lungs showed moderate impairment of resonance with some enfeeblement of the breath-sounds, but no râles. The chest-expansion was restricted but she experienced no noteworthy shortness of breath. I do not know her subsequent history but think I should have learned of it had her health not been good.

The fourth and last case of this series befell the wife of a brother practitioner in Chicago, Mrs. W. I was asked to see her because of a left-sided pleurisy from which she had been suffering for two or three weeks and which had followed what was thought to be an attack of grippe.

The lady was up and dressed but was very weak and short of breath. Her temperature was very slightly elevated above normal. The entire left side of the thorax was dull, though as in the preceding cases the note was only moderately so as far down as the third interspace in front and to a corresponding level behind. Below this point was flatness with marked feebleness of breath- and voice-sounds and with scarcely appreciable pectoral fremitus. In this case Traube's semilunar space could not be positively made out. The side did not bulge to any great degree.

The diagnosis seemed plain and on what appeared to be evidence of a considerable effusion, aspiration was advised. This was performed subsequently, the next day I believe, and the surgeon, a very competent one, was able to obtain only a few ounces of sero-fibrinous exudate.

My examination of this patient many months afterwards disclosed moderate loss of resonance and diminution of respiratory murmur over the lower portion of the left side, but her general health was excellent. She was in good health in the Summer of 1905. Whether this pleurisy was primary or secondary I know not, nor can I state its probable ætiology. In none of the cases was the fluid examined bacteriologically, much to my regret. Rosenbach states that under some circumstances the entire lung or a portion of a lung may be collapsed in consequence of a very moderate amount of effusion, and hence in the cases just narrated there may have been no pneumonia but a greater degree of collapse than was assumed at the time.

Purulent Pleurisy (Empyema).—The most of what has been said concerning the other forms of acute pleuritis applies also to this variety. The inflammation is usually secondary with the one important exception of pneumococcus empyema. In such instances the pleuritis coming on in the course of some general infection may be overlooked. But when it is secondary to some local disease (pulmonary abscess or gangrene, caries of a rib or vertebra, œsophageal cancer, perforation from the abdomen, etc.) the onset is sudden and characterized by pain and other symptoms that at once direct attention to the pleura.

In this class of cases the exudate if not actually putrid is nevertheless of so toxic a character that symptoms of profound intoxication soon set in. The pyrexia is high and continuous or shows marked ups and downs, and chills and sweatings may follow the oscillations in the temperature curve. The patients rapidly lose flesh and strength, become apathetic and pass into a typhoid or pyæmic state.

The sufferer lies on his back uttering no complaint; his lips and tongue are dry, his breath foul; the pulse is rapid and feeble but his breathing is not apt to evince dyspnœa; indeed it may surprise one to find so little evidence of distress from the local condition when, to judge from the physical signs, the purulent exudate is considerable. These cases require immediate evacuation of the pus if they are not to succumb.

Cases of pneumococcus empyema, on the other hand, are much more benign. In the first place the exudate is apt to be small and encysted and pressure-signs are insignificant or absent. The pus is not markedly virulent as is that of pyogenic cocci just described. It is so thick from admixture of fibrin that if the amount is small and it be left to take care of itself it may become inspissated from absorption of the fluid portion. Adhesions are then left but convalescence occurs in time.

It not rarely happens that the pneumococcus is mixed with some puscoccus and then the exudate is less benign. The amount of the pus is apt to be larger, it is not so likely to be circumscribed by adhesions, signs of pressure on, or strictly speaking displacement of, the heart are present, and symptoms of general poisoning are more or less apparent.

In the adult a pneumococcus empyema is most apt to develop during an acute fibrinous pneumonia and then is apt to be obscured or wholly concealed by the general and local symptoms of the pulmonary affection. In a paper on The Medical Aspects of Empyema published in 1893 I narrated the case of a gentleman who suffered for some weeks from this complication, the empyema being entirely unsuspected. As was common in such instances the persisting dullness in the postero-lateral aspect of the right base was taken to indicate an unresolved pneumonia.

Fever was so slight and strength had so far returned that the man was up and about his room. The one distressing symptom was cough which at times was violent, paroxysmal and prolonged. I saw him in consultation and from the history of a recent pneumonia, a careful study of the pectoral fremitus, the voice- and breath-sounds, I made a diagnosis of empyema. A surgeon operated soon thereafter and, according to my belief, accidentally punctured the lung; for, after having resected and discovered by palpation with his finger through the wound that he was too far to one side, he attempted to enter the pus-pocket by passing his instrument along between the two pleural surfaces.

His finger had detected a space between the costal pleura and the lung and somewhat farther toward the rear he had noticed a firm mass that filled the space at that point between the lung and chest-wall. It was this tumor which he endeavored to pierce from the side in order not to have to resect more rib. The moment his instrument penetrated this bulging mass, air escaped from the lung. On this ground the surgeon declared the condition was not empyema but a pulmonary abscess.

My contention was and still is that it was an encysted pneumococcus empyema because, by its presence, it had compressed the lung and thus caused what seemed to the surgeon's finger to be a space in front with a somewhat bulging mass toward the rear. Moreover, the thick, creamy pus, which fortunately had been saved, did not contain traces of pulmonary tissue. The patient ultimately made a good recovery.

The recognition of the true nature of these cases of small pneumococcus empyema is by no means easy at the bedside by the ordinary method of physical exploration of the chest. In all suspected cases, therefore, it is good practice to resort to exploratory puncture. More will be said about this means of diagnosis later on, but the statement may here be made that if strict asepsis be employed the dangers of this procedure are inferior to the harm likely to ensue from the failure to recognize a condition that calls for operative interference.

The development of pronounced sweating in a case of acute pleuritis with demonstrable effusion of some sort would naturally lead to the suspicion of its being purulent. Therefore, it may be well here to direct attention to the fact previously mentioned that severe, even colliquative, perspirations may be a symptom of pleurisy with sero-fibrinous and not purulent exudate.

Interlobar Empyema.—In not very frequent instances a pleuritic inflammation may be confined to the membrane lining an interlobar sulcus. This may be serous, but under such circumstances the process is not so likely to occasion marked general disturbance as when the exudate is purulent (interlobar empyema). Fowler states that since he has made it an invariable rule to scrutinize the pleura lining the interlobar fissures he has much more frequently recognized miliary tubercles as the cause of a primary serous pleurisy. This fact should be borne in mind in cases of interlobar collections of serum.

The general symptomatology of an interlobar empyema is not different from that of a purulent exudate in the free pleural cavity, but the local signs are often very obscure. It is to be remembered, moreover, that pus may occupy a sulcus between two lobes whereas the fluid in the general pleural sac is serous. Under such circumstances the evacuation of the serous exudate is not followed by complete subsidence of symptoms. The temperature may remain of a hectic type and the general condition may become very bad. The recognition of the interlobar pleurisy is impossible before aspiration, and even after this procedure it may be by conjecture rather than by positive signs that the interlobar empyema is diagnosed.

The following case in a twenty-months' old baby girl is instructive. The child was seen by me June 16, 1901. It was stated that the child had had measles in February previous, after which there had been an attack of bronchitis. In March she had had another illness called bronchitis, and since that attack she had been ailing with fever, some cough, restlessness, evident pain and had lost a little in weight. For the past two weeks the temperature had fluctuated from 98° or 99° F. in the morning to 102°, 103° and even 104° F. during afternoon or evening. Three days ago the temperature was low and the child perspired profusely. Three physicians had pronounced the child tuberculous and had administered tuberculin by mouth.

My examination disclosed the following: Respirations from 40 to 60 or even 80 per minute; the right half of the chest visibly larger than its fellow, the prominence being especially noticeable below the right clavicle; the upper abdominal zone also prominent and the respiratory movements of the liver visible; the respiratory movements of the right half of the chest visibly diminished as compared with those of the left lung; pectoral fremitus not diminished in front on the right but lessened in the axillary region. Percussion shows moderate dullness in right front as far down as the seventh costal cartilage; in the lateral aspect is more marked dullness and there is a slight but distinct sense of increased resistance; it is over this region that pectoral fremitus is diminished; auscultation perceives exaggerated, even bronchial, breathing in front, and diminished breath-sounds in the axillary region.

From the shape and lessened expansion of the right half of the thorax I was convinced of the existence of a pleuritic exudate which I suspected to be purulent and, from the long history, yet without more marked evidence of sepsis, to be of pneumococcus origin. After careful consideration of the situation of the dull area with diminution of fremitus in the lateral aspect, yet with bulging of the anterior aspect of the chest, I argued that the exudate was probably in the fissure between the upper and lower lobes and by its presence was pushing the lung outward in such a manner as to produce the appearance of the chest noted.

Accordingly, Dr. Bayard Holmes made an exploratory puncture in the very center of the lateral dull area (mid-axillary line opposite the nipple) and obtained thick pus. Resection of a rib then permitted access to the exudate which was found to occupy the interlobar fissure. Thick pus was evacuated, leaving a circular depression which by the finger could be perceived to be surrounded by a thickened pleura which prevented the lung from immediately expanding and closing up the sulcus. The child made an uninterrupted recovery.

Pulsating Empyema.—The peculiarity of this form of empyema lies in the production of a localized pulsation which, with very rare exceptions, is systolic, i. e., synchronous with cardiac contractions. Usually there is but a single area of pulsation though several such areas may be observed (Osler). The pulsation is ordinarily seen at the junction of the anterior with the lateral aspect of the chest, or indeed quite laterally, but according to J. G. Wilson and my own experience it may be observed in the back and lumbar region.

The conditions favoring pulsation are (1) great tension within the pleural sac as when it is completely filled by the exudate; (2) either necrosis of the costal pleura so that the pus lies in contact with the soft tissues of the parietes; or simple widening of the intercostal spaces and relaxation of the intercostal muscles. That a certain degree of tension is the chief factor is shown by the disappearance of the pulsation after removal of a few ounces of fluid. Comby's view that there must be compression of the lung and fixation of the heart by pericardial adhesions so that the cardiac impulse is communicated to both parietes and fluid, may apply in some instances but certainly not in all.

The area of pulsation which in consequence of the destruction of the costal pleura is also prominent is not extensive, 3 or 4 inches in diameter, and on this account may resemble the tumor caused by an intrathoracic aneurysm. The mistake of considering it an aneurysm is hardly likely to occur if one pay due regard to history and physical signs. In doubtful cases no harm would be likely to result from exploratory puncture with a very fine needle.

I recall the case of a child of some six or seven years whom I examined many years ago and who was suffering from a purulent exudate that completely filled the left pleural cavity. He was greatly emaciated so that the ribs and intercostal spaces could be easily distinguished. The latter were broad and yielding and the hand laid on the antero-lateral aspect of the chest on the affected side plainly distinguished a pulsation with each cardiac systole, though I do not recall any bulging in this region. In another child seen at St. Anthony's Hospital, Chicago, there was a pulsating tumor of the size of a large orange situated on the left back. This instance bears out Wilson's statements as to the posterior situation of some of these tumors.

Rupture of a pulsating empyema may take place, and when it does it is generally at the back, if the condition favoring perforation is necrosis (Osler); whereas without erosion of the soft parts rupture takes place anteriorly or laterally.

Pleurisy in children presents some symptomatic diversities from that in adults. In my experience the respirations are relatively more rapid and on excitement or other slight disturbing conditions become greatly increased. Consequently the respiration-rate is very variable. The pulse also is very frequent and often of unstable rhythm.

The degree and character of fever depend upon the kind of organism responsible for the pleurisy, in other words, upon the virulence of the inflammation, and hence upon the character of the exudate; and yet it should be borne in mind that children generally react with more intense pyrexia than do adults under similar conditions. The degree of pressure symptoms also depends upon the character and amount of the exudate. In some respects very young children seem to me remarkably tolerant of intrathoracic pressure, but if, as remarked by Rosenbach, the displacement of organs is less marked than in adults the congestion of the liver is certainly conspicuous, as evinced by distention of the upper abdominal zone.

It is in the course of pneumonia that one should be especially on the lookout for small pleuritic exudates in children. As is well known a pneumococcus pneumonia in these little patients is often of limited extent and hence it is easy to confound such a pneumonic patch with a circumscribed pleuritic exudate and the reverse. I have notes of a boy of six years who developed an acute pneumonia of pneumococcus origin on the left side in the lower part of the upper lobe, in the second interspace near the anterior axillary line. At the same time there was a somewhat larger area of dullness in the right infrascapular region below the inferior angle of the bone which yielded bronchial breathing and appeared to be a patch of consolidation. Pectoral fremitus was diminished, however, and an exploratory puncture obtained clear serous fluid. There was very little disturbance from either process in this case.

A girl of seven years was seen by me on account of fever which had lasted for a number of weeks and followed what was called by the family physician an attack of bronchitis. Cough was a marked feature at the time of my visit but there was no special dyspnœa. Over the left back corresponding accurately to the lower lobe was well-marked dullness with distinct bronchial breathing, no râles. The heart did not appear displaced, but probably was to some extent. The area of dullness did not shift with change of position of the patient. Fremitus was somewhat diminished yet not to the degree one would have expected in a pleuritic exudate.

Nevertheless, the diagnosis was made of an encysted empyema of pneumococcus origin, the nature of the infection being declared, I thought, by the fact of the exudate's being encysted. Operation was advised and upon resection of a rib and the incision of the intercostal tissues, thick odorless, somewhat opalescent pus actually spurted forth, so great had been its tension.

This case illustrates, I think, that if in young children there is not as marked displacement of the organs as in adults as stated by Rosenbach, it is because of the adhesions which serve to compress the exudate against the yielding lung. Only the area occupied by the exudate is compressed; the rest of the lung undergoes compensatory emphysema and thus serves to counterbalance the pull of the opposite lung. It is also possible that the pericardium is bound by adhesions in some cases.

Diaphragmatic Pleurisy.—Inflammation of the upper surface of this muscle or, to speak strictly, of the pleura overlying its upper surface, probably takes place to a greater or less extent in all cases of basic pleurisy. But by diaphragmatic pleuritis is designated a class of cases fortunately not often encountered, in which the process is limited to this region or predominates over that in other parts. It may be primary but is more often secondary to pneumonia or disease of some adjacent organ, e. g., subphrenic abscess, hepatic abscess or some other suppurative inflammation within the peritoneal cavity. In such cases the intercommunication between the lymph channels on the thoracic and abdominal aspects of the diaphragm facilitates extension of the inflammatory process.

The symptoms are very striking and suggestive. Pain is the most marked feature, is very intense and is felt in the epigastric region, in the hypochondrium or along the line of insertion of the diaphragm at the level of the tenth rib. It is so severe and so aggravated by movements of the muscle that the sufferer involuntarily breathes with the upper portion of the lung and restrains the excursion movements of the diaphragm. Accordingly, the type of breathing is costal. The countenance is anxious, denotes suffering and may be cyanosed. The pulse is rapid and often of a character to suggest dangerous cardiac weakness.

The patient generally sits up in bed in order, if possible, to restrict to the utmost the inspiratory descent of the diaphragm. He is often restless and changes position from time to time. He is alarmed and sure that he will not be able to endure his suffering and begs piteously for relief. In short the clinical picture is one of far greater distress than is seen in most cases of acute pleurisy.

The inflammation may lead to the exudation of lymph merely (dry pleurisy), or a more extensive exudate may form which may be sero-fibrinous or, in cases secondary to some abdominal affection, purulent. The quantity is not apt to be large and yet is sufficient to cause cessation or mitigation of the pain and possibly to yield signs of its presence.

The inflammation of the diaphragmatic pleura leads to fixation but not paralysis of the muscle (Fowler) and hence the respiratory movements of the affected half of the chest are likely to be practically abolished, so that the hypochondrium may be motionless or appear flattened in distinct contrast to what is usually seen. A friction rub is likely to be absent or possibly may be detected very low down along the inferior margin of the affected lung. The recognition of the condition must depend on the symptoms rather than on the local findings, and hence must be rather by exclusion than by positive signs.

Physical Signs. Inspection.—The phenomena discernible to the eye differ somewhat in the two forms of pleurisy, i. e., the dry and the effusive. In the initial stage of the process the respiratory movements on the affected side are restricted or partially inhibited on account of the pain caused by inspiration. There is, consequently, an apparent flattening of the lower and anterolateral aspect of the diseased half. The upper zone and the opposite half of the thorax may show an exaggeration of respiratory play. The patient may lean somewhat to the inflamed side and thus intensify the apparent decrease in the diameters of the side.

Litten's sign is diminished or abolished on the diseased side. This sign is a shadow seen at the line of insertion of the diaphragm on either side, near the outer margin of the anterior aspect, which shadow moves downward on inspiration and upward on expiration and is caused by the alternate descent and ascent of the diaphragm. In order to detect Litten's shadow sign the patient must lie on his back in a strong light and the observer must stand at a little distance so as to look obliquely on to the surface of the chest.

With *accumulation of fluid* in the sac the signs of its presence obtained by inspection differ somewhat from those just mentioned. The affected half of the thorax now appears increased in size and the intercostal spaces are visibly widened, filled out and even bulged, according to the amount of exudate. The respiratory movements are also diminished or retarded as compared with those of the opposite side. To perceive this increase in the diameters of the side the examiner should view the chest from various angles and not solely from the front.

If the apex-beat is visible it is seen displaced to a variable extent toward the opposite side. In young subjects the upper segment of the abdomen may appear distended and the liver may be seen to project somewhat and to move up and down with the acts of respiration. The displacement and congestion of the organ are better perceived by palpation, however.

In very extreme cases the enlargement of the affected half of the chest is very conspicuous and the spinal column may deviate from a straight line, the convexity being toward the distended half.

Pressure effects may also appear in the turgescence of the cutaneous veins and possible œdema of the skin, in the epigastric pulsation, in the dyspnœa, etc., though in these latter there is nothing peculiar to fluid accumulation in the pleural sac.

Palpation.—In the commencement of the inflammation and in the plastic variety, palpation may yield negative results. Exceptionally, however, I have known friction fremitus to be palpable over a limited area—this in thin-walled chests. Palpation is of service in the determination of the presence or absence of visceral displacement.

With accumulation of much fluid, palpation enables one to appreciate the enlargement in the diameters of the side, and the widening and prominence or the smoothing out of the intercostal spaces. In exceptional instances fluctuation may be appreciated in the tense broadened interspaces. Vocal fremitus is diminished or abolished over the region occupied by the fluid.

Mensuration.—This is of value in the accurate determination of differences in the size of the two halves of the chest. The tape may be used for this purpose and care should be exercised to mark the median line front and back by a pencil. As the object of such measurement is to obtain the relative diameter of the two halves and not the expansibility of the chest, the tape should be read during expiration or the suspension of breathing. The cyrtometer is an excellent means of noting any inequality in the two halves. This is a strip of lead preferably covered with leather which, after having been accurately molded around one side of the chest, is removed and laid on a sheet of paper. Its outline is then traced with a pencil and the contour thus obtained is compared with that of the other side after this has been measured in the same manner and at the same level. This requires care and patience but is a method of obtaining precise comparisons which is declared by those who are familiar with it to be of great value. I have not employed it to any extent and never in cases of intrapleural accumulations. *A priori* cyrtometry ought to be a reliable means of determining increase and decrease in the amount of effusion.

Percussion.—This is a very reliable means of recognizing the presence of a pleuritic exudate. In the initial stage of pleurisy, however, and in the dry form percussion yields but negative results. That is, the note over the inflamed lung is still resonant or at the most very slightly impaired.

As the effusion takes place this is declared by dullness which, beginning at the bottom of the affected side, extends upward to a variable distance. Directly above the level of the fluid there may be tympany. In any suspected case the examiner should carefully outline the upper limit of dullness since this has certain definite characters.

In the first place this upper margin of dullness possesses a curvilinear outline known as the *S-shaped line of Ellis.* Beginning at the spinal column (Fig. 98) it passes upward and outward reaching its highest point in the axillary region. From here the dullness passes forward and somewhat downward until on the anterior surface of the chest-wall it again ascends slightly to the sternum. In small effusions this peculiar S-shape is not distinguishable,

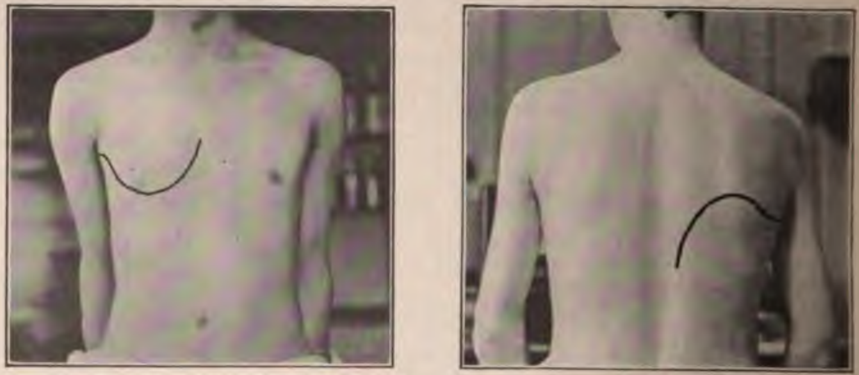


Fig. 98.—Outline of "S"-shaped line of Ellis in large pleuritic effusions.

the line of dullness curving upward from the spine and downward to the base of the pleural cavity, as when a scanty exudate occupies a limited region at the posterior and inferior part of the chest (Fig. 99).

In the second place, the line of dullness will generally be found to alter its direction in accordance with posture. Accordingly, when there is a limited zone of dullness at the base behind, it will often be found that if the patient leans forward the upper margin of dullness occupies a lower level than in the erect posture. If one tests this change in the level of the fluid in front it will be perceived that dullness extends higher when the patient sits than when he reclines; or one may test the mobility of the exudate by having the patient roll from one side to the other while percussion is performed. It goes without saying that if the exudate be encysted, as is so often the case in a pneumococcus pleurisy, this alteration in the level of the exudate, i. e., the upper line of dullness, does not take place. Nor is the mobility of the exudate always so apparent when this is purulent as when it is serous.

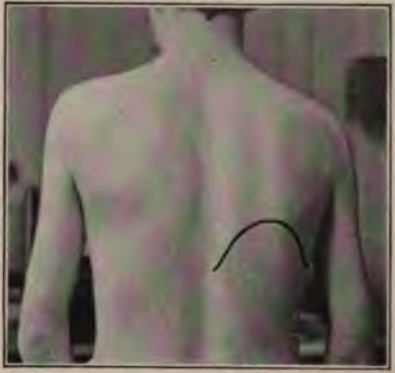


FIG. 99.—Line of dullness in smaller effusions.

When in massive pleuritic effusions the fluid reaches to the level of the third rib in front or perhaps higher, it will be perceived that the percussion-note immediately above the area of flatness possesses a high-pitched tympanitic quality (skodaic resonance). It not infrequently happens that the note over the apex is more or less dull, though never so flat as is that over the fluid itself. There is also a sense of increased resistance on percussion over the portion of the thoracic cavity occupied by the exudate.

Auscultatory percussion is a means of recognizing the presence of a pleuritic effusion which has often proved of service to me. It is performed by tapping the chest gently with the palmar surface of the middle finger while by means of a stethoscope auscultation is done at some point within the area of dullness. As long as one taps above the level of the fluid he hears the note distantly and feebly, but as soon as the exudate is reached the note becomes clear cut and sharp since the vibrations are conducted plainly to the ear. It is often useful to shift the position of the stethoscope, placing it now above the line of dullness, now below, while at the same time percussion is made toward the supposed exudate in various lines of direction. By auscultating now over the liver, now over the exudate and now over the lung, one can often accurately determine the limits of the exudate. I have found this of great service before deciding where to perform aspiration.

Percussion reveals resonance often exaggerated, over the healthy lung and it is of aid many times in ascertaining the position of the heart when this cannot be determined by inspection or palpation. It also enables one to detect displacement of the liver.

It is often stated that in left-sided effusions percussion reveals dullness instead of tympany in *Traube's semilunar space*. This is a space occupying the lower segment of the chest immediately to the left of the epigastrium. If a line be drawn from the inner extremity of the seventh left costal cartilage (Fig. 100) outward for a distance of about three inches and then sharply downward to the inferior costal margin it will inclose a space within which the percussion-note is tympanitic from the presence of the stomach beneath.

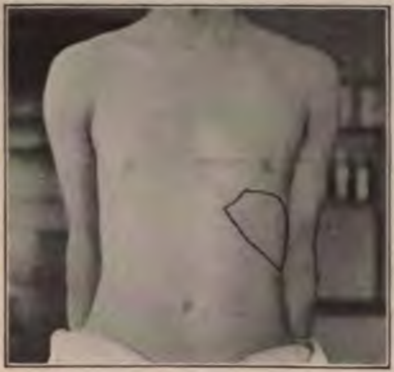


FIG. 100.—Semilunar space of Traube.

That dullness may replace tympany in this region in some cases of extensive serous effusion on the left side cannot be denied. But I believe it is also true that obliteration of Traube's space is not always produced. At all events it is not always easy to decide whether in some cases this space is or is not preserved.

Auscultation.—In the initial stage of acute pleurisy and in most cases of dry pleurisy is to be heard the so-called pleuritic *friction sound* or *pleuritic rub*. This is a succession of fine crackling sounds which are produced by the separation of the two pleural surfaces or by their rubbing together when rendered sticky and adherent by the coating of lymph exuded. Its characters are not the same in all cases. It may and usually does give one the impression of two sticky surfaces being forcibly torn apart and is then made up of a succession of minute râles generated close to the ear. At other times it has a creaking quality like the noise occasioned by the rubbing of new leather or the creak of a saddle, while in other cases it may have a parchmentlike quality. Whatever its timbre it is generally heard with both acts of respiration, though not exclusively so, being in some instances limited to inspiration. I believe also that I have heard it during expiration alone.

This friction rub is usually most distinct with the first one or two deep inspirations and afterwards it may greatly diminish or even disappear. It should be carefully auscultated for, in different areas throughout the entire side and not merely at the seat of pain, since this may be referred to some point quite distant from the seat of the exudate. Often it is extremely localized and unless carefully sought for may escape detection.

The breath-sounds at this time are usually diminished and may be almost or quite obscured by the friction râles. The voice-sounds may also be enfeebled.

In *sero-fibrinous effusion* the friction rub generally fails or is audible very indistinctly and imperfectly, but this is not without exception. In such cases the friction-sound may be inaudible over the center of the dull area and yet be heard along its upper margin. In small empyemata I have often noted an obscure and indefinite sound that was hardly distinguishable as a râle, but gave the impression of a muffled rub as if two wet blotting-papers were being passed over each other. This indefinite sound was always appreciable along the margin of the zone. A rub may sometimes be inaudible in one posture but become audible when the change of position causes the exudate to gravitate to another level.

Coincident with the accumulation of fluid the breath- and voice-sounds become enfeebled, as a rule, or may assume a distant bronchial quality. In children the breathing throughout the dull area may be loud and bronchial. These cases may then be hard to differentiate from a pneumonic consolidation. I have seen cases in adults in which, the breath-sounds being bronchial and the dullness immobile and râles absent, they could not without puncture be distinguished from a pneumonia.

Auscultation is of aid also in determining the position of the heart. Breath-sounds over the opposite lung are likely to be exaggerated and, in some instances, there may be bronchitic râles. Ægophony on which stress used to be laid in the recognition of a small effusion I do not now recall having ever heard, although I have sought for it repeatedly and diligently.

Diagnosis.—The recognition of acute pleurisy is not difficult ordinarily. The frank onset, the pain in the side, cough, fever and accelerated breathing constitute a complexus of symptoms in themselves suggestive of the affection. If on examination of the lungs the characteristic rub is detected, no doubt of its existence can be entertained. If dullness does not supervene or if this is at the best but very slight and circumscribed, the condition may very properly be set down as a dry or fibrinous pleuritis.

In a sero-fibrinous or purulent pleurisy with considerable exudate the problem is likewise not difficult of solution. As before, there are the history and general symptoms and possibly also the friction-sound, but if the case be seen for the first time after effusion has formed and the rub is no longer present, then considerable difficulty may arise. In all instances, therefore, especial attention must be given to the detection of the physical signs already described.

These are: (*a*) enlargement of the base or of the entire half of the thorax; (*b*) widening and filling out of the intercostal spaces at the base, most apparent in the lateral aspect; (*c*) diminution or absence of tactile fremitus over a limited portion or the major part of the side; (*d*) dullness situated at the base, having a curved upper line as previously described, which is usually found to change in position according to posture; (*e*) diminished or absent breath-sounds those audible being broncho-vesicular, bronchial or indeterminate; (*f*) possible friction along the upper margin of the exudate or, on change of posture, becoming audible in areas where before it was absent owing to the interposition of the exudate; (*g*) displacement of the heart toward the opposite side, shown ordinarily by location of the apex-beat; (*h*) downward displacement of the liver; (*i*) obliteration or diminution of Traube's space, in left-sided effusions; (*j*) skodaic resonance above the level of massive effusions and below the clavicle.

Difficulty is encountered in cases of small and possibly encysted exudates, e. g., a small pneumococcus empyema or an interlobar pleurisy. In such cases particular notice must be taken of the state of pectoral fremitus, the characteristics of the dull area and the signs of pressure. If the tactile fremitus be carefully studied it may sometimes be noticed to be absent or almost abolished over the center of the dull patch and to be plainly appreciable nearer to and at the circumference.

In small exudates the shape of the upper line of dullness is not so useful as is any change in its level. This may be and often is so slight as to be rather indefinite; yet by using the dermatographic pencil and carefully marking the line in varying postures one can usually form a tolerably clear opinion on this important point. Visceral displacement is also a highly valuable aid, but unfortunately this is either absent or inappreciable in cases of small exudates in the lateral regions. In all cases of doubtful diagnosis I would urge a trial of auscultatory percussion after the manner previously described.

Exploratory Puncture as an Aid to Diagnosis.—If one rely on physical examination of the chest the nature of the great majority of cases will become clear, and yet every now and then some cases will be found so puzzling as to necessitate resort to some other means for diagnosis. This additional means is exploratory puncture with a hypodermic needle. I agree fully with Rosenbach in his recommendation of the ordinary hypodermic syringe for this purpose instead of an aspirating needle, and yet the latter in the hands of the expert is perfectly reliable. Every general practitioner carries a small hypodermic syringe in his pocket, and if he select a long needle and the parietes be not unusually thick he will find it an easy matter to penetrate the pleural sac.

Certain precautions are necessary. The needle should be carefully sterilized by boiling. The skin of the part to be explored and the hands of the operator must be scrubbed with soap and water and afterwards with alcohol or bichloride solution. The needle should be inserted as nearly as possible in the center of greatest dullness and if possible where the musculature is not very heavy. The exploration should be made in the same posture in which dullness was detected; as will be seen in the remarks on aspiration in the consideration of treatment it is very possible for an exudate to be missed if the position of the patient be not noted and be not kept the same as when dullness was demonstrated.

Some of the precautions insisted upon by Rosenbach are so pertinent that I shall reproduce them in effect here. Certain conditions may be responsible for the failure to obtain fluid. The exudate may be thick and rich in flocculi; if, in such an event, the needle be fine its point may become plugged by a drop of pus or a flake of fibrin. Under these circumstances no fluid may be seen to flow into the barrel of the syringe or the piston may spring back, thus forcing the obstructing substance back into the pleural sac. Therefore, whenever exudate does not appear to be present Rosenbach advises that the syringe be withdrawn without allowing the piston to fly back and that any possible obstructing material be afterward expressed onto a clean cover-glass. Any drop of pus that may be extruded can then be examined by means of the microscope or by the unaided eye.

The failure to obtain fluid in one area should not be considered final, but the needle should be introduced in several spots; for it is very possible for exudate to be missed at one point on account of adhesions and yet found at another near by. Moreover, one pocket may contain serum and in another not far distant there may be pus. This statement leads naturally to another; namely, the only reliable means of determining the *nature* of an exudate is by exploratory puncture. It is at the same time the simplest. If local anæsthesia be secured by a spray of ethyl chloride or by the application of a mixture of salt and ice, pain from the puncture can be avoided. Still, even delicate patients will be found to bear well the slight shock of an exploratory puncture with a needle of small caliber.

The notion seems to prevail, although probably not so widely as formerly, that exploratory puncture is liable to infect a serous exudate and convert it into a purulent one. I will not deny such a possibility if the operator is dirty. But if he carefully sterilize his needle, the field of operation and his own hands there is small danger of infecting the pleural cavity. I hope no reader of these lines will be guilty of carefully disinfecting his needle and then of laying it down on the bed, as was done by a surgeon in my presence.

Lastly an exploratory puncture is of value many times in determining whether an exudate is sero-fibrinous or purulent and in enabling the physician to ascertain the ætiology of the pleuritis. If the specific gravity be high it furnishes a certain amount of evidence in favor of a tuberculous or carcinomatous origin. If, on the other hand, it be rich in fibrin the exudate is probably of pneumococcus origin. Again, an exudate rich in polymorphonuclear leucocytes is due to the pneumonia germ or to pus-cocci; whereas a predominance of lymphocytes is held to indicate a primary tuberculous pleurisy. In a pleuritis secondary to tuberculosis of the lung, the fluid may contain an excess of the polymorphonuclear elements. If the exudate is due to malignant disease there may be cells characteristic of the growth or a preponderance of epithelial cells. These last also abound in transudates. Furthermore, if the fluid be allowed to stand and the sediment be centrifuged, properly stained and examined microscopically, it is sometimes possible to discover bacilli or other bacteria. Lastly, in tuberculous or cancerous pleurisies, the serum may be bloody. In endothelioma of the pleura, the needle obtains dark-colored blood (Fraenkel).

The X-ray as an Aid to Diagnosis.—The recognition of pleuritic effusions may be made by this means but it is rarely necessary to resort to the X-ray in cases of considerable exudate unless perchance conditions exist which prevent one from arriving at a positive conclusion otherwise, or an exploratory puncture is impossible or also negative. The fluorescent screen is preferable to a photograph (Williams). It is particularly of service in the recognition of obscure exudates as interlobar empyema. In such a case there would be seen a shadow occupying the position of the exudate, provided, of course, the surrounding lung were at all clear. The screen enables one to perceive mobility of the effusion, displacement of the heart and obliteration of the diaphragm line (Williams). The curving upper surface of the fluid should also be apparent and this might enable one to differentiate the condition from obscure cases of pneumonia or to determine if the latter affection coexist with the pleuritis.

The **diagnosis of diaphragmatic pleurisy** is also said to be facilitated by the use of the fluorescent screen but only, as I conceive, when effusion has taken place. Its recognition is not then so important as when, in the acute early stage, there are the alarming symptoms previously noted. This may be inferred when the intensity of the suffering, the cyanosis, the air-hunger, the præcordial pain and the action of the heart all are out of proportion to the physical evidence of pleuritic inflammation. Hiccough is very suggestive and if with these pronounced symptoms a friction-sound can nowhere be detected the presence of diaphragmatic pleurisy is probable.

The **diagnosis of an interlobar pleurisy** is attended with great perplexity especially in young children. The local signs are most pronounced or confined to some point along the line of an interlobar fissure. Potain, who has bestowed much attention upon this form of pleurisy, attaches special value to careful study of tactile fremitus and of the voice-sounds. Should a circumscribed empyema of this location be associated with a sero-fibrinous effusion of the general pleural cavity, as sometimes happens, the diagnosis of the former is possible only after aspiration of the latter, and then will be improbable in the majority of instances.

Differential Diagnosis.—In the first place it is necessary to differentiate the pleuritic friction-sound from other râles of similar character. Unquestionably there come times when even the experienced auscultator is in doubt as to the nature of the adventitious sounds. In general, however, the recognition of its true character is not difficult. The two kinds of râles which simulate a rub are subcrepitant mucous clicks in pneumonia and bronchitis and the fine bubbling sounds of pulmonary œdema at the base. If the râles are altered in their characteristics by cough they are not pleuritic. If the rub has a scraping, creaking or distinctly scratching quality it cannot be mistaken. Friction often sounds as if generated on the surface and close to the ear. In most instances it is heard with both inspiration and expiration, though I am convinced that exceptions do occur.

In dry pleuritis the detection of this friction rub makes the diagnosis so plain that errors cannot occur. Yet for the very reason that the rub may fail it is possible to confuse myalgia and intercostal neuralgia with this form of pleuritis. In most instances there is no need of mistaking these latter affections for acute dry pleurisy and yet this is precisely what is done by careless practitioners.

In *myalgia* there is apt to be a history of exposure to a draught after getting into a perspiration, or of strain of the muscles, or of injury, as a contusion. The muscle is found to be sensitive and painful when pinched or when caused to contract by some movement calling the muscle into play. Cough is absent unless having preëxisted and constitutional symptoms pointing to pleurisy are wanting. A friction rub is not present.

Intercostal neuralgia like the foregoing may closely simulate dry pleuritis by causing pain in the side on deep inspiration, sneezing, coughing, etc. In the first place, a friction rub and fever are absent. There are usually definite tender points along the line of certain nerves. The breathing may be superficial but is not often accelerated as in pleurisy. But in all such cases chief dependence must be placed on the absence of any other symptoms or signs such as are a part of the clinical picture of pleurisy.

Acute Pneumonia.—When dullness exists the differential diagnosis must be made between pleurisy and pneumonia. In the great majority of instances there is no great difficulty in this matter or the physician recognizes that both conditions exist. The points in favor of pneumonia are the following: (1) The disease begins in practically all cases with a distinct rigor; whereas in pleuritis the chill, if present at all, is less pronounced or is repeated. (2) Dullness does not present the same outline, does not change on posture, does not increase from above downward. If in a small patch the dullness does not reach to the spine or is separated from the lower limit of the lung by a zone of comparative resonance, it is probably consolidation and not pleural exudate. The dullness is not apt to be so intense as in pleurisy. (3) Theoretically, pectoral fremitus should be increased or at least unimpaired over a pneumonia, whereas in pleurisy with effusion it is decreased or absent. In practice, however, one often finds cases of pneumonia in which, owing to secretions in the tubes or to coincident fibrinous pleurisy, the tactile fremitus is diminished. (4) In pneumonia the breath-sounds are audible, bronchial and most plainly so over the area of greatest dullness. It may also be that a few crepitant râles will be caught at the close of deep inspiration which, of course, would settle the matter. In effusion the breath-sounds are of lessened intensity and possibly absent, at all events are not so typically bronchial as in pneumonia. (5) Signs of displacement of adjacent organs are not present or are not so apparent as in effusion and the intercostal spaces do not feel so smoothed out. If, after exhausting these various means of investigation, the nature of the case is still doubtful, resort may be had to exploratory puncture.

The difficulties in the way of correct diagnosis and the value of puncture are well illustrated by the case of Mr. W., reported in Differential Diagnosis of Pneumonia, in which old adhesions so bound down the right lung as to prevent displacement of heart and liver. The location of the adhesions was such, moreover, that the exudate was forced to occupy a relatively high level and compress the entire lung more or less; so that the dullness presented no upward curved line and did not shift its position with change in the patient's posture. Had an exploratory puncture been consented to it would have corrected our mistake and perhaps have saved the man's life.

Malignant disease of the lung is hardly likely to be confounded with acute pleurisy so long as the latter is not produced by the cancer. Still, it is possible that a primary growth of large size might, if occupying the lower lobe, simulate a pleural exudate sufficiently to mislead at first. The area and other characters of the dullness will be different, pressure-effects will be seen not to correspond to those of intrapleural fluid and the general symptoms will be unlike, so that the differentiation ought to be possible. If pleuritic effusion be produced by the neoplasm it is likely to obscure the growth and may be mistaken for a primary pleuritis. For details see chapter on Malignant Growths in the Lung.

The points in the diagnosis of *echinococcus cyst* and of *actinomycosis* of the lung will be found in the respective chapters and need not be repeated here.

Hepatic abscess and a *subphrenic abscess*, particularly the latter, may, under some circumstances, occasion perplexity in their differentiation from pleurisy with effusion. Due attention to history, symptoms and physical findings should enable one to recognize their true nature. In the latter, special study must be given to the breath- and voice-sounds and to tactile fremitus, all of which are absent over the seat of the subdiaphragmatic collection of pus.

For the differentiation of a pleuritic exudate from a transudate, see the diagnosis of hydrothorax.

Complications.—Acute pleuritis may be complicated by inflammatory processes in other organs, e. g., pneumonia, pericarditis. As regards the former, objection may be made to its being called a complication on the ground that both affections may be due to the same cause and that the pleurisy is rather a complication of the pneumonia. Nevertheless, cases are seen now and then in which the pleurisy seems to precede and which would doubtless not prove a very serious affair were not pneumonia also present. The latter may affect the same or the opposite lung.

The existence of pericardial inflammation adds a very grave complication to a pleurisy, and if the sac be much distended it, as well as the pleural cavity, should be emptied to prevent the heart from being disastrously overburdened.

Fortunately the modern practice of tapping pleural effusions, and in particular empyemas, renders far less likely the complication of necrosis of the external parietes and spontaneous evacuation in this direction. Treatment of such a condition is surgical. Evacuation of an encysted empyema through the bronchi may, in some instances, prove a bad complication and prolong the duration, but may be relieved by the surgeon.

Some of the symptoms may at times be so troublesome as to prove veritable complications. Thus there may be an obstinate and almost incessant cough, or bedsores may result and the nutrition of the skin become seriously impaired by the prolonged decubitus acting in conjunction with pyrexia. Congestion of the liver may prove very painful and the disturbance of circulation in the digestive organs may unfavorably affect appetite and the power of digestion, so that the strength of the patient may be maintained with difficulty. A symptomatic nephritis may occasion the physician much uneasiness, yet in ordinary instances this will not materially influence prognosis. A femoral vein may become thrombosed as in any other acute infection and increase the patient's discomfort but it is not usually a grave complication.

For my part, I have never seen any especially serious complications although I have known persons to die from a pleuritis.

The COURSE of an acute pleuritis depends upon the severity of the process, and upon whether or not it leads to extensive exudate. An ordinary dry pleuritis is likely to terminate in from a few days to two or three weeks. A sero-fibrinous effusion, if left to itself, may terminate by resorption of the inflammatory products in from three to six weeks. On the other hand, it may run on into a chronic affair. Purulent pleurisies, if not operated upon, either destroy life in a few weeks or pass on into a chronic condition which months later shows up as a pulsating empyema or *empyema necessitatis.* Encysted pneumococcus empyemata may run a very chronic course and eventually become absorbed or inspissated.

Prognosis.—This depends upon various factors but, as a rule, is favorable to recovery. Fibrinous pleurisies are usually not very formidable illnesses and a return to health may be predicted. There may be left, however, adhesions of the two layers of the pleura with each other, or of the pleura with the pericardium, of so extensive a sort as materially to impair vital capacity or cardiac function. An extensive dry pleurisy may ultimately lead to considerable retraction of the side.

The outlook in extensive exudates depends upon the degree of pressure exerted and also upon the rapidity with which they form. Great tolerance may be established to a slowly accumulating effusion even when this ultimately fills the whole pleural cavity. *Per contra* a comparatively small exudate may, by its rapid collection, seriously compromise breathing and the individual's powers of endurance.

Purulent pleuritis is always a serious matter and if not recognized and operated upon is more likely to terminate in death than recovery. No time, therefore, should be lost in resorting to aspiration or resection as soon as the complaint has been identified. The rapid development of chills, fever of a hectic type and sweatings renders prognosis extremely grave and calls for immediate surgical intervention.

The occurrence of profound constitutional disturbance in any form of exudate is to be regarded as serious. Pleuritis in the old, in the debilitated or alcoholic, in connection with chronic nephritis or cardiac disease, and in the obese is always to be looked upon as a serious disease. The occurrence of complications adds to the gravity in proportion to the kind and severity of the complication.

Unless acute exudative pleuritis is properly treated it must be expected to leave behind adhesions of more or less serious moment. In fact, only when the effusion is very largely serous and without much if any fibrin can adhesions not be expected to result. Consequently, the effect on the future health will be determined by the amount of fibrin deposited upon the pleural surfaces.

Primary tuberculous pleurisies do not inevitably leave the individual in impaired health, yet the fact that so large a percentage of such pleurisies are followed in subsequent years by pulmonary tuberculosis is a matter not to be forgotten in the subsequent management of the case.

Pneumococcus pleurisies are relatively very benign even when purulent, but they are particularly likely to be succeeded by extensive and dense adhesions.

Streptococcus pleurisies and all others caused by pus-cocci are the most serious and produce the most profound symptoms. Putrid exudates in particular are very dangerous if not speedily operated upon. A hæmorrhagic pleurisy does not in itself indicate a grave prognosis independent of the disease that may underlie it.

Treatment. EARLY OR ACUTE STAGE.—Since acute pleuritis is a manifestation of infection of the pleural sac we have no means of cutting short the inflammation. We may modify the intensity of the process and mitigate the severity of symptoms, but we cannot antagonize the action of the germ to the extent of speedily arresting the pathological process. Nevertheless, I do not advocate a spirit of therapeutic nihilism, for experience teaches that certain measures exert some modifying influence in cases in which the *causus morbi* is not very virulent.

Medicinal Agencies.—The experience of Dock in this country and of Fiedler, Rosenbach and Aufrecht abroad indicates that *salicylate of soda* diminishes the intensity of the symptoms if administered in the commencement of the affection. It is especially indicated in the formative stage of the exudate when fever is high and pain pronounced. It is of no service after the effusion has formed and become stationary. The rationale of this treatment is based on the experiments of Rosenbach and Pohl which showed that salicylates make their way into the pleural cavity whenever this is occupied by a fluid either exudate or transudate. On this basis it is believed that they may restrain the action of the organisms concerned in the production of the inflammation. The dose recommended is from 4 to 8 grammes daily (ʒj to ʒij) for an adult.

Antipyretic Remedies.—The coal-tar derivatives, in particular, are not indicated and are likely to do more harm than good. On the other hand, it is good practice to relieve the pain and promote sleep by the administration of an opiate, and the most efficacious is a hypodermic of morphine. The dose should be large enough to quiet and not merely stimulate the sufferer; accordingly, ¼ to ⅛ of a grain is better than ⅛, but the physician must be governed by his own judgment in this matter. Children may be given a bromide and in some instances this may be reënforced by a small dose of codeine or even a hypodermic. My experience convinces me that in children of four years and more morphine is not harmful if employed with care and judgment, for relief of pain.

If cough happens to be a troublesome symptom, aggravating the pain and robbing the patient of sleep, it should be relieved. To this end heroin $\frac{1}{12}$ to ⅙ grain may be prescribed or morphine may be given hypodermically. Heroin deepens the respirations and on this account may increase the pain. In most instances the treatment addressed to amelioration of pain does precisely what is indicated by the cough, and further measures are not necessary.

Local applications are sometimes of positive advantage in this acute stage. If the temperature is high and the patient not very feeble I prefer the application of an *ice-bag* to the affected side. This may be kept in place as long as it is grateful to the patient. Should it occasion annoyance or prove depressing it may give way to moist heat in form of a poultice, or to a mustard plaster. In some instances wet cups are beneficial but I have never employed them, nor indeed blood-letting in any form in this malady.

Strapping the side is in my experience very serviceable for relief of pain, provided it is done properly. Adhesive straps must be at least two inches in width, must be laid on smoothly and firmly *during forced expiration* since their purpose is to restrict the expansion of the painful side. They should begin just beyond the median line behind and terminate past the median line in front. There should be two layers of the strips crossing each other as shown in Fig. 101. In young children whose chests are easily compressed a broad cloth bandage drawn tightly about the thorax at the base may answer. It is not as effective, however, as proper strapping.

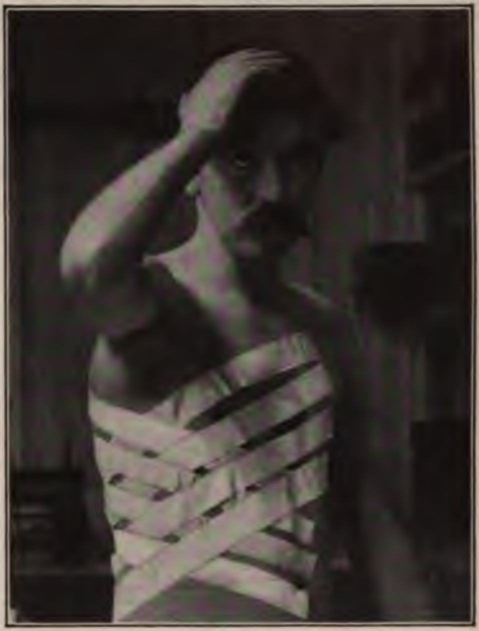


FIG. 101.—Method of application of adhesive straps in pleuritis.

I have been told by one of my medical friends that he has seen marked relief of pain afforded by massage of the intercostal spaces in which the suffering is felt. The massage is performed by stroking the respective point or points firmly with the tips of the fingers and continuing this stroking for five or more minutes, until pain is mitigated. I cannot name the person who deserves the credit of this discovery but believe it originated in Sweden.

The **nourishment** in this stage should be such as can be best digested by a febrile patient; milk, broths, soups, raw eggs, gruels, milk-toast, wine or lemon jelly, etc. Whether or not the dietary should be exclusively fluid is to be determined by the height of fever and other conditions. It is better when not too hearty or stimulating.

The state of the bowels and the action of the kidneys must be carefully watched. If opiates have to be administered or if, for any other reason, the action of the bowels is sluggish a *laxative* should be ordered, e. g., citrate of magnesia or a dose of salts or some aperient water. Calomel is useful in many cases and, in fact, I believe it is always well to initiate the treatment of every case of acute infectious malady with a dose of this excellent drug. *Water*, weak lemonade or other fruit juices in solution, should be administered freely to these patients for the purpose of promoting diuresis and thus endeavoring to eliminate toxins.

STAGE OF EFFUSION.—As soon as the exudate appears to have reached its limit, i. e., is no longer increasing, it should be removed either by aspiration or, if possible, by some measures calculated to further absorption. Not only should the height of the effusion be carefully ascertained by examination of the chest each day, for several successive days, but the symptoms should be carefully studied. It may usually be considered that disappearance or marked decrease of fever indicates a diminution in the inflammatory process (of course I am now speaking of sero-fibrinous effusions and not of purulent exudates). In the latter case the pyrexia is likely to continue and bespeaks septicæmia and not inflammation.

Diuretics are, in my opinion, of very doubtful service in promoting absorption of the fluid. In some cases spontaneous absorption begins after a time and hence we cannot say positively that the effect attributed to medicinal treatment is not, in reality, a manifestation of nature's efforts in this direction. I agree with Osler, however, and many other men of experience in the opinion that it is good practice to order a daily morning dose of some saline cathartic. The particular one selected is not important; it may be sulphate of sodium or magnesium or some of the aperient waters, but the dose should be such as to cause several loose stools. Painting the surface of the chest with iodine is, according to my belief, of no service in promoting absorption, notwithstanding the recommendation of many authorities.

Aspiration.—This procedure is a distinct advance over the old-time plan of letting an effusion remain for weeks in the hope of its being spontaneously absorbed. Francis Delafield made a study of cases of sero-fibrinous effusion treated by early evacuation of the fluid and came to the conclusion that cases so treated did better than those not so managed. Theoretically also he is right in his conclusion; for it appeals to me as probable that if an effusion is left *in situ* for a term of weeks the fibrin constituents are certain to be deposited on the pleuræ in larger or smaller amount. Subsequent organization of the deposit is likely to take place and serious retraction of the side and permanent collapse of the lung will ensue.

It is my belief also that one should not delay this operation too long. As soon as the physician is certain that the exudate is not increasing and as soon as the decrease in the violence of the symptoms points to cessation of the active inflammation aspiration should be performed. On the other hand it is harmful to operate in the beginning of the exudative stage. Moreover, it is not necessary nor is it well to remove as much of the fluid as possible. Years ago Roberts advocated the withdrawal of only a portion of the exudate on the ground that subsequent absorption will be promoted by the diminution of pressure incident to the removal of only a part of the fluid. I believe this rule is still a good one to follow and I am pleased to see that Rosenbach concurs in this opinion. He advocates the removal of about one half the amount.

Many practitioners make it a rule to abstract fluid until the patient begins to cough or complain of pain. It seems to me that the operation should cease before these symptoms appear. It appeals to me as possible that the removal of all or nearly all the fluid favors a reaccumulation of the exudate if pain is experienced. It indicates, moreover, that the active inflammation of the membrane has not yet disappeared and that it would have been better to leave enough fluid in to prevent the membranes from coming into contact with each other.

Moreover, it is not unlikely that the sudden removal of the whole amount favors the production of pulmonary œdema. At all events this is a catastrophe the possible occurrence of which should be borne in mind. Whatever be the cause of acute pulmonary œdema in these cases, it is not considered good practice to remove the fluid rapidly.

The rapid withdrawal of a large effusion from about a collapsed lung carries in its train the rapid reëxpansion of the organ and this must tend to its congestion. This may be announced by cough which is more or less severe in proportion to the degree of congestion and the lung's ability to respond to the suddenly altered conditions. Under some circumstances this may prove a very serious affair and hence should be guarded against by the precautions above advocated.

The expectoration of a profuse albuminous fluid, which is termed *acute pulmonary œdema,* fortunately does not follow many cases of thoracentesis, but we are never in position to affirm it will not ensue. Whether, as advocated by Leichtenstern, it is owing to some defect in the nutrition of the blood-vessels or, as argued by Rosenbach, it is owing to escape of lymph in consequence of abnormal widening of the interstices in the walls of the vessels, it is an expression of acute congestion. Hence, although it may not always be prevented it is the more liable to occur the more rapidly the exudate is removed.

For these reasons the use of the aspirator should be cautious and the fluid be allowed to run out of its own free will, so to speak; and in the beginning of the operation, at least, it should not be assisted by too energetic manipulation of the pump. If the above precautions be duly considered it is not probable that harm will result.

The failure of spontaneous absorption does not furnish the sole indication for aspiration. Occasionally an exudate forms with such rapidity as seriously to threaten life, danger being shown by grave circulatory and respiratory embarrassment. In such an event one cannot wait for the effusion to become stationary, but must resort to aspiration without unnecessary delay. Here again, however, I would urge that only so much of the fluid be withdrawn as will afford the needed relief. The operation may be repeated at a later time as occasion may require.

Finally, I desire to direct attention to the circumstance that it is quite possible to have a large serous effusion and yet fail to obtain fluid if the patient be placed in such a posture as facilitates gravitation of the fluid away from the site of puncture.

A striking proof of this was afforded by the following case: I was requested by a physician to see his father who was suffering from symptoms of cardiac incompetence. To make a long story short I may state that the heart was being overtaxed by a pleuritic effusion which had formed insidiously and had not been recognized. The right pleural sac was half full of movable exudate, as shown by the results of percussion.

Accordingly, the physician, who was not present at the time of my visit, was informed of my finding and advised to have it removed. The next day I was notified that a surgeon had explored in five places and failed to find fluid. Being very sure of my diagnosis I revisited the patient and demonstrated the effusion by physical examination. I then inquired as to his posture at the time of the puncture and was told he had been lying down. Placing the man in a sitting position I then inserted the needle of my hypodermic syringe and withdrew a syringeful of clear serum. Upon such ocular evidence of the presence of the exudate the surgeon was again summoned. Presumably the aspiration was performed with the patient in the erect posture, for 80 ounces were removed.

The occurrence of slight cough after the operation may be ignored. But if this prove severe and beyond the patient's ability to restrain it is very likely to favor pulmonary congestion. Therefore, it will be well to administer a hypodermic of morphine, whereupon the likelihood of harm will be averted.

In many instances the aspiration will not be succeeded by return of fluid, provided the operation has been postponed until active inflammation has subsided; or it will not recur in such an amount as necessitates a repetition of the procedure. Nevertheless, should this recur or should the residue of the exudate left *in situ* fail to undergo absorption, then a resort to one or more aspirations at not too short intervals is justifiable.

The Operative Treatment of Empyema.—It goes without saying that surgical intervention is the only plan of management advocated in this class of cases. A pneumococcus empyema may be aspirated in some cases and the patient recover. Should symptoms not abate resection should be done. In all other cases, or rather in cases the symptoms of which show a virulent exudate, rib-resection is indicated in order that permanent drainage may be secured. The question of an immediate operation as soon as exploratory puncture has demonstrated the nature of the case must be left for answer to circumstances. I am in the habit of advising no unnecessary delay in these cases. The methods of operating will not be described in this work as they are described in text-books on surgery.

The after-treatment of cases that have been operated upon offers no difficulties in most instances, and presents no problems meriting special consideration.

It may be added, however, that I see no indication for the administration of diuretics, diaphoretics, digitalis, acetate of potash, etc., for the purpose of preventing return of the exudate. Modern views of the pathology of acute pleurisy make it clear that if inflammation subsides and the exudate has not been left too long *in situ* it is not likely to recur; or if it does, we have no means in medicinal agents of preventing it.

Tonics of various kinds, as iron, arsenic, hypophosphites and even oil, may sometimes be indicated. But this must be left to the judgment of the attending physician. Life out of doors or change of climate may be of great benefit in some cases. In particular the general health should be built up when the disease is believed to be of tuberculous origin. Complications and subsequent intercostal neuralgias will be met as they arise. Breathing and other exercises to prevent contractions are indicated and should be insisted upon as important.

CHAPTER XXXVI

CHRONIC INFLAMMATION OF THE PLEURA

Ætiology. Adhesive Form.—The presence of adhesions between the two pleural membranes or between these and neighboring parts, i. e., the mediastinum, is an exceedingly common *post-mortem* finding. In some instances there may be a history of acute pleurisy or of acute pneumonia which accounts for the existence of the adhesions (Figs. 102–103). In such a case the changes in the pleuræ are the remains of an inflammatory process that has long ceased to show any activity whatever, and yet they are usually placed in the category of chronic pleuritis (Plate XII).

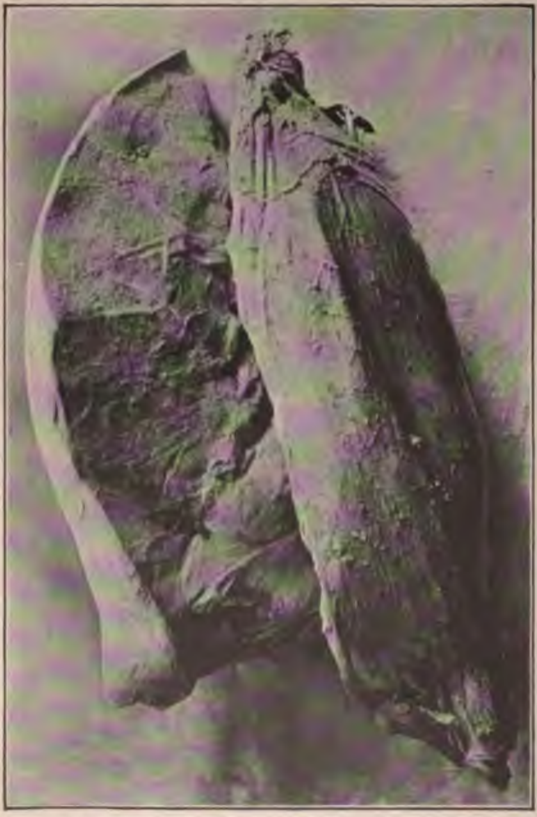


FIG. 102.—Chronic fibrous thickening of the pleura.

In other cases the fibrous bands uniting the pleural surfaces are really the result of a proliferative process that has been going for years. It is probable that some of these instances have their starting-point in an acute though not very active process. They are, therefore, examples of insidious or *latent pleurisy*. The precise ætiology of these cannot be stated but some of them may be tuberculous. In some instances there is good reason to believe that they are of syphilitic origin.

There are other cases in which the obliterative process going on in the pleural cavity is but a part of a general tendency to chronic insidious inflammation of serous structures (a general serositis). Instances of this kind are seen in persons who were subjects of chronic proliferative mediastino-pericarditis, which condition may or may not have been diagnosed *intra vitam.* A case of the kind described in my work on Diseases of the Heart and Arterial System concerned a Mrs. M. The symptoms occasioned by the chronic pleurisy were wholly subordinate to those produced by the development of fibrous tissue in and about the liver (ascites), as well as those referable to the mitral disease from which she had long been known to suffer.

Nevertheless, many months before her death this lady developed fine crackling friction râles along the anterior base of her right lung which gradually spread in extent until at length they could be heard as far up as the infraclavicular region and in the lateral aspect of the side. They were audible with inspiration, and for a long time puzzled me considerably. In the light of the autopsy findings it was clear that the râles were the auscultatory evidence of a slowly progressing inflammation of the pleuræ. In this instance the peritonæum showed the same obliterative inflammation.

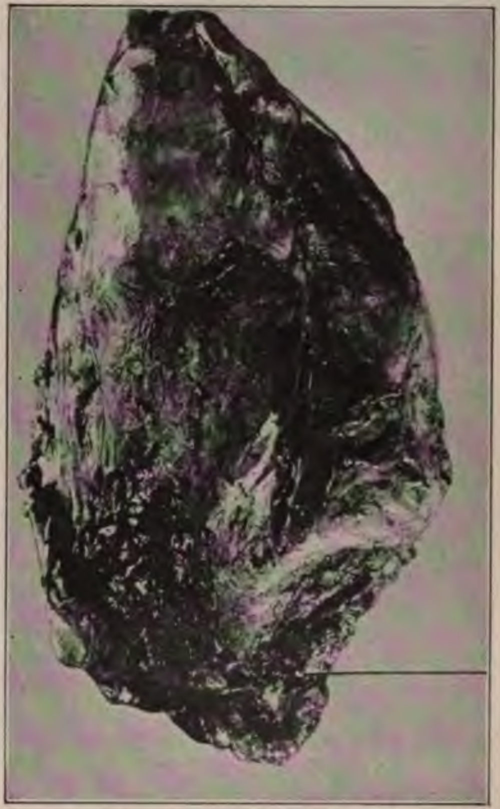


FIG. 103.—Chronic fibrous thickening of the pleura. Line indicates pocket occupied by a small sacculated empyema.

Sero-fibrinous Form.—This variety, characterized by the formation of a more or less abundant sero-fibrinous effusion, may, like the fibrous variety, be the outgrowth, i. e., the extension of an acute frank inflammation or it may develop insidiously. In the former the effusion fails merely to be absorbed, and that is about all one can say about it. In the other set of cases the inflammatory process sneaks in, so to speak, like a thief in the night, without making its presence felt. Symptoms do not assert themselves early and force the individual to consult a physician. Consequently, the presence of the effusion is only discovered after it has become fully established, or it is chanced upon in the *post-mortem* room.

The precise nature of these latent sero-fibrinous effusions is a matter for conjecture, but they are probably tuberculous in most instances. When they are discovered in the old or in persons with cardio-vascular and renal disease it is not always easy to decide whether they really are exudative or transudative; particularly if the fluid be on the right side and very poor in fibrin.

Morbid Anatomy.—Chronic pleurisy displays itself as old adhesions, as a persisting sero-fibrinous effusion or as a pyothorax. In the first form there may be but a few loose fibrous bands at the base or these may be distributed widely throughout the pleural cavity. In some cases the sac is completely obliterated by a layer of organized lymph and the lung cannot be removed without being lacerated. In these instances the proliferative process has invaded the connective tissue of the lung and the organ presents the changes described under the term cirrhosis of the lung. The organ is more or less shrunken either in whole or in part and shows bronchiectases. The side of the chest is correspondingly retracted and deformed (Fig. 37). Not infrequently circumscribed adhesions are found at the apex and cover over a zone which, from the presence of old scars or calcareous nodules, is plainly tuberculous.

In the sero-fibrinous form of chronic pleuritis the membrane generally shows more or less thickening and, in some parts, even bands of fibrous tissue. The lung is collapsed to a degree commensurate with the amount of fluid in the sac (Fig. 44). In some instances of not very long standing, it is plain that only the presence of the fluid has kept the lung from expanding, for after removal from the body it is still capable of inflation. At other times the organ is fibrous and in a condition to which the term carnification has been applied. Neighboring viscera are found displaced and the right ventricle is dilated and hypertrophied.

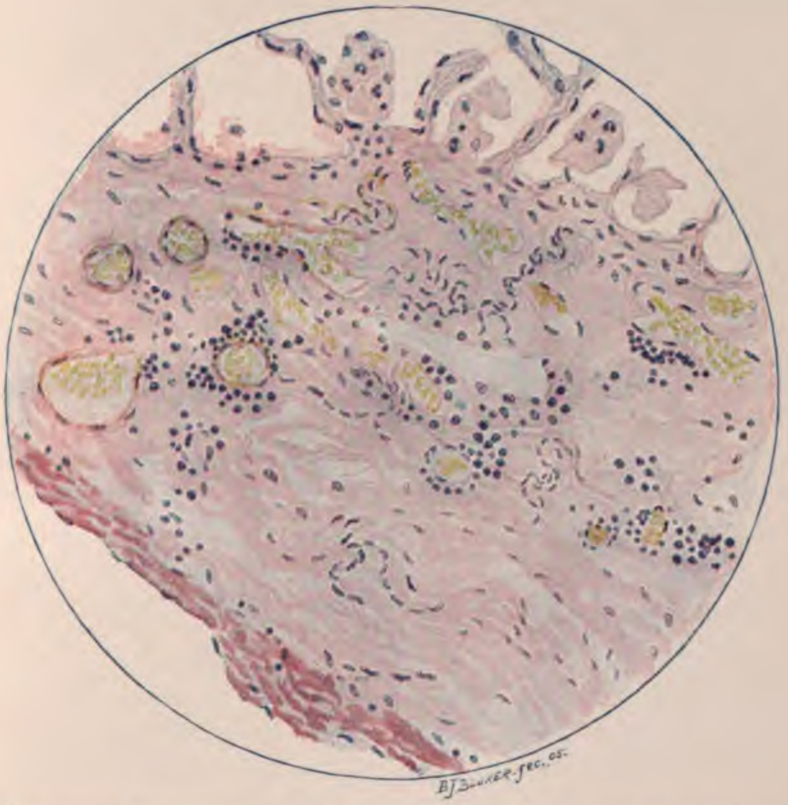
If a chronic pyothorax (empyema) is the condition, it is generally in connection with chronic pulmonary tuberculosis. It may be in other cases of pneumococcus origin, when it is likely to be encysted and possibly interlobar. If the empyema is tuberculous it is very apt to have led to the formation of a sinus and to have discharged externally (*empyema necessitatis*). I do not now recall having ever seen such an instance in this country but very plainly recall one at the polyclinic in Munich in 1882. The production of a pulsating empyema has already been dealt with in the preceding chapter and to it the reader is referred.

In such a case there are collapse of the lung and the displacement of the heart and abdominal organs, as in the sero-fibrinous form.

Symptoms.—From what was said under morbid anatomy it is evident that chronic fibrinous pleurisy may or may not occasion symptoms according to the extent and firmness of the adhesions. In the great majority of cases their presence is not suspected, or, at the most, they serve only to impair pulmonary resonance at one base and to produce more or less distinct friction in the same area on deep inspiration. The excursion movement of the diaphragm may be restricted or abolished, as shown by the absence of Litten's sign.

In other cases the adhesions may lead to serious disturbance of circulation in consequence of permanent displacement of the heart or great blood-vessels: or they occasion cirrhosis of the lung and corresponding deformity

PLATE XII



Microscopic Section through Pleuritic Exudate Undergoing Organization into Firm Fibrous Tissue.

of that half of the thorax. The symptoms are then such as are met with in that affection, namely, cough, expectoration and dyspnœa. If bronchiectasis results the symptoms are those that have been described in that chapter.

Persons who have recovered from an attack of acute pleurisy or from an acute croupous pneumonia not infrequently complain of pain in the side that was affected, which pain is apt to be worse in damp and changeable weather. In some of these instances the presence of adhesions may be determined from the restricted respiratory movement or from the absence of Litten's shadow sign, as stated above. The pain in these cases is of the nature of an intercostal neuralgia and appears to depend in some way on the adhesions. It generally wears away in the course of time.

Chronic sero-fibrinous pleuritis impairs the health sooner or later even when it does not occasion dyspnœa, cough and cardiac feebleness, as the direct effects of the fluid. In some of these cases it is remarkable how little embarrassment is occasioned, aside from the effect on the general health.

Thus, I recall an instance seen when I was a student in New York. The man presented himself at the clinic with the history of a pleurisy many months before and complained of his inability to work at his occupation of street-car conductor. Examination disclosed signs of fluid that completely filled the right pleural sac, distention of the side, entire absence of pectoral fremitus, dullness nay flatness throughout, and want of breath-sounds save at the apex. Thoracentesis was subsequently performed and something more than a gallon of clear serum was obtained. The lung did not reëxpand and the fluid re-collected.

Such a procedure is not justifiable since the fibrosis of the collapsed lung prevents the reëstablishment of its function. The condition being incurable the victim of a chronic pleurisy of this kind drags out a miserable existence and ultimately succumbs to the pressure-effects or to some intercurrent affection to which he is predisposed by reason of the long existing pleurisy.

Physical Signs.—These are essentially such as were described under acute pleurisy and do not need to be recapitulated *in extenso.*

Inspection.—The most reliable and, one might say, the only evidence of pleural adhesions furnished by inspection is the absence of the Litten shadow sign. The descent of the diaphragm may be hindered or abolished even when the expansion of the lung in general is not perceptibly affected. When the adhesions are at the apex their presence may be shown by failure of the upper lobe to expand synchronously or equally with its fellow. Such a state of things is usually taken to indicate pulmonary tuberculosis, and hence great attention must be paid to other signs and symptoms before one can conclude definitely that merely adhesions exist.

If a chronic sero-fibrinous effusion is present the affected half of the chest is perceptibly greater in its diameters than is its fellow. There is also the ocular evidence of displacement of the heart's apex. There may be, in addition, more or less dilatation of superficial veins.

Palpation.—This is negative in the case of adhesions, but in fluid distention of the pleural sac pectoral fremitus is diminished or abolished. In cases of pulmonary fibrosis due to chronic obliterative pleurisy the presence of extensive adhesions may be suspected if tactile fremitus is wholly wanting at the base.

Percussion.—Circumscribed adhesions do not of necessity alter the percussion-note, but if they are dense and extensive the affected lung is likely to be found dull either in areas or quite generally about the base. It is observed also that the excursion movements of the inferior margin of the lung are decreased or even abolished. If fluid is present the note is flat and the dull area displays the characters described in the acute form.

Auscultation.—There may be no auscultatory evidence of circumscribed adhesions. The breath-sounds may be somewhat diminished, it is true, or, if the thickening of the pleura is sufficient to compress slightly the underlying lung, there may be broncho-vesicular breathing. But this is often too indefinite to be really appreciable. A friction-sound of more or less distinctness may be heard in some instances, but unless the rub is very characteristic the examiner is likely to be uncertain of what he really does hear.

In a considerable number of persons of middle or old age deep inspiration is attended at the bases with a number of fine dry clicks that sound very like a friction produced by the rubbing together of two dry membranes. In some cases these may be pleural but in others they are due to the expansion of partly collapsed lobules. Similarly, one sometimes hears in the young over the upper part of the chest, indefinite crackling sounds that suggest friction, yet which are in reality muscular and due to the contraction of the muscles as the lung is forcibly inflated. Again, Broadbent has recently described a friction-sound produced in the pleura and also in the pericardium, apparently in consequence of the administration of iodides, since the rub disappeared after discontinuance of the remedy.

Whether his theory of its being produced by an artificial dryness of the membrane be correct or not, this phenomenon as well as the facts just stated suggest the wisdom of great reserve in always regarding as evidence of old adhesions the frictionlike sounds often heard in doubtful cases.

If a chronic sero-fibrinous pleurisy be present the breath-sounds are likely to be greatly diminished or, indeed, wholly absent. Such is apt to be the case over the inferior parts of the affected half. At the apex, on the other hand, the breath-sounds may be audible and are then harsh or bronchial, especially at the back, in consequence of the crowding of the lung upward and backward. Voice-sounds are likewise greatly enfeebled or absent.

Diagnosis.—From the foregoing it is apparent that the existence of pleural adhesions is a matter largely of conjecture in many instances. In some cases they may be reasonably assumed from the retraction of the side and the diminution in the respiratory movements of the lung, or from the absence of Litten's shadow sign. Unmistakable friction-rub over a limited zone would, together with a history of previous pleurisy or pneumonia, be diagnostic, but without such a history it must be taken with great reserve in the light of Broadbent's observations.

Decreased respiratory expansion of a lung, impaired resonance at the base, diminished tactile fremitus and enfeebled breath-sounds would, all taken together, furnish tolerably good evidence of the existence of old and extensive adhesions. If the affected half of the chest is considerably retracted and flat there can be scarcely any doubt of fibrous thickening of the pleura. Yet the case previously mentioned as seen by me in the Munich hospital, in which these signs were exhibited and exploratory puncture yielded blood, and which was thought to be one of malignant disease, shows how difficult may be the diagnosis of pleural thickening in some instances.

The diagnosis of a chronic effusion ought to be made with ease and certainty in most cases from the physical signs. In doubtful cases exploratory puncture or the X-ray will settle the diagnosis. The differentiation of such a fluid from a transudate is to be made by chemical analysis; see Hydrothorax.

Prognosis.—This depends upon the nature and extent of the process. Circumscribed adhesions do not necessarily affect the prospect of life. Dense fibrous thickening of the pleura does, on the other hand, seriously affect the health and hence will probably shorten life materially. The more serious the secondary changes the worse is the prognosis.

A sero-fibrinous exudate that does not undergo absorption is a very grave matter, not necessarily in the near future, but from the fact that it can scarcely fail to impair general health even when its pressure-effects are in themselves not serious. The prognosis is grave, moreover, because of our inability to remedy the condition surgically or otherwise after it has existed for many months. The inability of the lung to expand after removal of the fluid is sufficient proof of the gravity of the case. Death is not likely to come from the effect of the fluid directly, but from the ultimate effect on the general health and from some intercurrent affection.

Treatment.—Circumscribed pleural adhesions that produce no obvious symptoms require no treatment. When a case is encountered which displays plain evidence of extensive, firm, fibrous bands and in which symptoms are complained of, we possess no means of causing absorption of the adhesions. They have become organized and obliterate the pleural cavity. We are limited to treatment of symptoms and must do what we can to improve the general health and to prolong life. In some cases the chronic pleurisy is wholly subordinate to the pulmonary fibrosis and atelectasis and hence the management is that appropriate to those conditions.

If the pleurisy is circumscribed and is thought of tuberculous origin, the measures indicated are such as are appropriate to pulmonary tuberculosis. Whatever be the precise ætiology of such limited pleurisies it is well early in the process to institute a system of respiratory gymnastics with a view to preventing contraction of the adhesions and ultimate decrease in the capacity of the lung. I am persuaded of the utility of such exercises provided the pleurisy is of comparatively recent date. When, however, retraction of the side has once begun to show itself there is considerable doubt of much being accomplished by any system of breathing exercises, unless it be in the way of producing compensatory emphysema of the sound lung.

A chronic sero-fibrinous pleurisy is a very perplexing proposition. In the first place one may reasonably inquire what constitutes a chronic case of this kind. In reply it may be said that when any case of recent pleuritis with effusion has persisted for several weeks and all symptoms of active inflammation have disappeared, yet the fluid remains, it has become chronic. It is presumed that aspiration has been resorted to, several times perhaps, and that the fluid reaccumulates after each evacuation. What is now to be done?

Just here comes the difficulty! For my part, I am frank to confess that the outlook is not brilliant. Nevertheless, there are several procedures that may be tried. If repeated tappings seem to be followed by the return of less and less fluid, then the operations should be repeated in the hope of eventual closure of the sac by adhesions and cessation of the fluid. It is recommended in some of these cases that incision and permanent drainage be established in the hope of ultimate obliteration of the pleural cavity. I have also seen it advised that in intractable effusions the evacuation of the exudate be followed by the injection into the sac of an equal quantity of sterilized water containing chloride of sodium in the proper proportion to constitute the normal physiological salt solution in the hope that, by maintaining the same degree of intrapleural pressure as before, an exudate cannot recur and ultimate absorption of the artificial serum may ensue. Although I have recommended this procedure in cases seen in consultation I have received no subsequent reports from the attending physicians and therefore know not how it has succeeded, if indeed it has been tried.

It is also stated that the re-formation of the exudate may be prevented by the injection into the pleural sac of a drachm of a 1 to 1,000 solution of adrenalin immediately after the withdrawal of the exudate. The procedure is simple but how efficient I cannot state from experience. Theoretically I should regard it as of no use in cases in which the fluid has shown no tendency to diminution after repeated tappings. It seems to me that incision and drainage would then be preferable.

The results of drainage or of oft-repeated aspiration must depend largely on the ability of the lung to reëxpand. Consequently, if the lung has undergone carnification, the only hope for the patient lies in the obliteration of the sac by organized fibrin and great retraction of the thoracic wall. If the fluid has remained for many months it is, in my opinion, doubtful if incision and drainage would result in obliteration of the pleural cavity; and if the fluid were not occasioning great discomfort or seriously affecting the general health I should advise leaving it alone.

CHAPTER XXXVII

HYDROPS PLEURÆ

I. HYDROTHORAX

THIS is a transudation and not an exudation of serous fluid into the pleural cavity and cannot be regarded as a distinct clinical entity. It is a symptom but one that sometimes proves so important in its pressure-effects as to make its consideration here advisable.

Ætiology.—Hydrothorax occurs as a local expression of general dropsy resulting either from hydræmia or from venous stasis. In either event, but particularly in the former case, it is apt to be associated with œdema of the lower extremities and with ascites. Under these circumstances the hydrothorax is usually bilateral, although it is common for one pleural sac, generally the right, to contain more fluid than does the other.

The diseases which lead to transudation into the pleural cavities are most frequently renal or cardiac, though not exclusively so. Nephritis occasions a hydræmia and this condition favors general dropsy. But not all forms of nephritis lead equally to transudations, it being well known that anasarca develops most rapidly and extensively in acute and chronic parenchymatous nephritis and in amyloid degeneration of the kidney when there is great cachexia. In chronic interstitial nephritis, subcutaneous dropsy and transudations into the serous cavities are seen only in the terminal stage, and then chiefly when grave cardiac asthenia has become superadded. In this condition there is a combination of hydræmia with venous stasis. In these cases there is also a retention in the system of the chloride of sodium, as shown by its elimination in the urine.

In heart disease of whatever chronic form, but especially in mitral disease, hydrothorax is a frequent observation and, strangely enough, it is apt to be unilateral and more often on the right than on the left side. Why this is, we cannot say, unless it be dependent in some way upon a peculiarity of the return circulation from the right pleural cavity which favors the occurrence of stasis. There are certain clinical as well as experimental facts that go to prove that venous stasis, either general or local, is not the only factor in the production of dropsy. Thus, we may see one patient with most serious cardiac inadequacy, possibly even with tricuspid insufficiency, and yet without œdema; whereas another, with a far less evident embarrassment of the circulation develops dropsy with transudation into all the serous cavities. We now know that an increased permeability of the capillary walls is even more important a cause of œdema than is venous stasis. In most cases, therefore, hydræmia and stasis are combined, though one may predominate as in the cachexias of amyloid disease and in acute parenchymatous nephritis.

Cirrhosis of the liver is another disease which, in its terminal stage, may be attended with hydrothorax. Inasmuch, however, as hepatic cirrhosis may be complicated by pleuritis of tuberculous origin we cannot always determine whether the intrapleural fluid is a transudate or an exudate; and, indeed, it is possible for both conditions to coexist in the one serous cavity, the inflammatory process becoming superadded as a result of irritation. Finally, hydrothorax may be secondary to any intrathoracic disease, as mediastinal tumor or aneurysm, which leads to local stasis.

Morbid Anatomy.—The pathological anatomical changes discovered in cases of hydrothorax really belong to the primary affection. The pleural surface may have lost its luster and in some places there may be old adhesions. When such is the case, however, they belong to some inflammatory process of long before and form no part of the transudation *per se*. The lung is more or less collapsed but is capable of reëxpansion. If the hydrothorax is unilateral there is some degree of cardiac displacement. On section of the lung it is generally more or less œdematous and displays brown induration. Such changes as are found in other viscera depend upon the original disease and are not at all the effect of the transudate.

Symptoms.—These are in large measure such as form the clinical picture of the chronic cardiac or renal disease. The presence of the fluid in one or both pleural cavities undoubtedly intensifies the dyspnœa that existed previously. In some instances, on the other hand, the shortness of breath amounting even to orthopnœa is really due to the hydrothorax which has led to collapse of some part of the lung. This is shown by the circumstance that if the transudate be drawn off by tapping, the patient feels much relieved and may even be enabled to recover some degree of health. The hydrothorax is generally associated with more or less pulmonary œdema and hence there is apt to be troublesome cough with frothy or blood-tinged sputum.

Physical Signs.—In many cases the transudation of serum into the pleural sac is only discovered after it has become of considerable amount, and, since it is so difficult to decide how much of the symptomatology is attributable to the hydrothorax and how much to the primary disease, the symptoms of the condition appear to be more objective than subjective. The presence of the transudate is apt to be concealed in part by the coexisting dropsy of other tissues.

Inspection.—When the fluid distention of the thoracic cavity is considerable inspection may discover some increase in the diameter of one or even both halves of the chest. But often this enlargement, if it exists, is disguised by the fullness of the abdomen caused by hepatic stasis and general visceral engorgement or by the ascitic distention of the belly.

Palpation.—The interposition of the transudate between lung and chest-wall prevents the conduction of vocal vibrations to the parietes, and hence tactile fremitus is diminished or wanting over the base of the chest occupied by the fluid.

Percussion.—This affords the best means of recognizing the existence of hydrothorax. The note over the base of one or both lungs is dull to a degree commensurate with the amount of the fluid. Often it is but slightly impaired and the examiner is in doubt whether the dullness be any more than can be justly attributed to pulmonary congestion or to the œdema of the overlying soft tissues. In all cases, therefore, the effect of change of posture should be tested. In hydrothorax this is usually very pronounced since a transudate in a serous cavity gravitates readily from one level to another.

It is stated that the upper line of dullness due to hydrothorax is straighter than that of a serous exudate. But I have never been able to satisfy myself of the correctness of this statement. Indeed, I see no good reason why this should be so; for the upward curving line of dullness in pleurisy with effusion is due to the corresponding curve in the surface of the diaphragm, and this is not necessarily lost whenever a transudate is present.

Auscultation.—If the hydrothorax is considerable it interferes with the transmission of the breath-sounds and hence these are enfeebled or absent. As a matter of experience the respiratory murmur is very apt to be obscured by the moist râles incident to the congestion and œdema of the bases or, when râles do not exist, the stasis within the lungs may yet cause feebleness of the breath-sounds; so that auscultation is of less aid in the detection of hydrothorax than is percussion.

Diagnosis.—The recognition of the presence of fluid in the pleural sac is ordinarily not difficult, resting, as it does, upon absence of percussion-resonance over the lower portion of the chest, together with loss of tactile fremitus and feebleness or entire absence of the breath-sounds. The upper line of dullness is said to be more nearly straight than is that of a pleuritic exudate; but, to my mind, this statement is scarcely borne out by clinical experience. The determination of the mobility of the superior line of dullness corresponding to posture is very important and proves beyond question that fluid of some kind exists.

Given signs of fluid in one or both pleural cavities in association with anasarca or ascites in a person suffering from some chronic disease likely to occasion dropsy, there cannot ordinarily be any doubt of the existence of hydrothorax. There are cases sometimes in which much perplexity arises, as will be seen in the differential diagnosis below.

It may happen when general dropsy exists that the physician is in doubt whether or not the dullness at the base of the chest is due to upward pressure of the liver in consequence of the ascitic distention of the abdomen; or the high degree of pulmonary congestion and the collapse of the lung render the lungs more or less impaired and the observer is not quite certain whether or not there is sufficient dullness at the base to signify the presence of fluid. Under such circumstances one may have to make an exploratory puncture or await the result of treatment and repeated examinations. It has been my experience that a brisk purge would sometimes lessen pulmonary stasis sufficiently to render pulmonary resonance a little more clear and thus bring out more definitely the line of demarcation between it and the dullness due to the fluid. Then by testing the mobility of the dull area I have been able to satisfy myself of the existence of fluid, without having to resort to the aspirating needle.

Differential Diagnosis.—When doubt arises in the mind of the clinician it is not as to the presence of intrapleural fluid or of some consolidation of the lung from any one of a variety of causes, but pertains to the question whether the fluid is a transudate or an exudate. The statements of the text-books lead one to conclude that the determination of this question is easy if reliance be placed on a chemical examination of the fluid removed by aspiration. Thus, it is generally stated that the specific gravity and the albumin-percentage are both lower in transudates than in exudates.

Such is undoubtedly the case and quite naturally so for the reason that the chemical features of a transudate approach more nearly those of the blood-serum. Nevertheless, the figures given for a pleural transudate and those given for a pleuritic exudate are found to overlap somewhat, so as to make the question not so simple as might at first be thought.

In a very valuable paper entitled Transudates and Exudates with reports on 75 Fluids, which appeared in American Medicine November 12, 1904, J. L. Miller has collected figures and statements which are most instructive and helpful in the determination of the nature of an intrapleural fluid.

For the sake of convenience I have annexed a table which is a part of one given by Miller.

	Specific Gravity.		Albumin.	
	Maximum.	Minimum.	Maximum.	Minimum.
Chronic nephritis (hydræmia):				
Neuenkirchen, 36 cases	1.010	1.005	....	....
Bernheim	1.010	1.005	....	....
Runeberg	....	....	.41	.03
Citron	1.010	1.008	....	....
Miller (pleura, 4)	1.009	1.008	....	....
" (abdomen, 2)	1.010	1.009	....	....
General venous stasis:				
Neuenkirchen (pleura, 21)	1.016	1.007	....	....
" (abdomen)	1.014	1.006	....	....
Runeberg (abdomen)	....	....	2.3	.84
Citron	1.016	1.009	....	....
Cirrhosis of liver:				
Neuenkirchen, 36 cases	1.014	1.006	....	....
Runeberg, 79 cases	....	....	2.68	.37
Bernheim	1.016	1.006	3.45	.56
Citron	1.016	1.008	....	....
Ott	1.022	1.006	6.88	.56
Miller, 20 cases	1.017	1.010	....	....
Pleuritis:				
Neuenkirchen, 29 cases	1.022	1.014	....	....
Runeberg, 27 cases	....	....	6.04	4.78
Ott	1.026	1.009	8.9	2.4
Miller, 27 cases	1.025	1.014	....	....

From a study of this table it is seen that a hydrothorax occurring in the hydræmia of nephritis may be quite accurately differentiated from a sero-fibrinous exudate, whereas one due to venous stasis may not be so readily distinguished in every instance since the maximum figures of the one overlap the minimum figures of the other. In such an event, therefore, aid must be sought in a careful study of the anamnesis and symptoms.

The following case illustrates the value of a study of the specific gravity and albumin-percentage in enabling one to arrive at a diagnosis and, in a measure at least, at the prognosis. S., a blacksmith, German, aged thirty-one, was referred to me September 7, 1905, by Dr. J. H. Rogers of Michigan City. The history was rather meager, being, in brief, that of an illness which began in December, 1904, with pain in the back that came on after lifting a heavy weight. What was his initial illness, or for what his first physician treated him, was not clear. When Dr. Rogers took him in hand he was dropsical and his condition was believed to be due to heart disease. Months of treatment failed to produce appreciable improvement, and when the patient consulted me he was in a pitiable state of weakness, dyspnœa and what seemed to be general dropsy.

The face, upper extremities and chest were much emaciated and noticeably pale, the pallor being very like that of nephritis. The lower portion of the thorax, the abdomen and the lower extremities were enormously swollen, in striking contrast to the wasted appearance of the upper part of the body. It required but a very brief examination to determine that the swelling of the legs and thighs was due to œdema which made the skin so tense that it could be pitted only by firm pressure. The peritonæum, both pleural cavities and, I am inclined to believe, the pericardium also were greatly distended with fluid.

The pulse was regular, very feeble and 120, while the heart-sounds were scarcely audible and the entire front of the chest was dull, so that the cardiac area could not be distinguished from the dullness due to the intrapleural fluid. Respirations were accelerated and shallow, but cough and expectoration were not admitted. The temperature was normal. The urine was scanty, showed a specific gravity of 1.010, a trace of albumin but no casts.

At first sight it seemed as if the condition was one of general dropsy secondary to chronic cardio-renal disease, and yet I gained the impression of some condition other than a primary cardiac inadequacy as responsible for the man's dropsy. Accordingly, he was ordered to a hospital for further study.

The next day 6 ounces of fluid were withdrawn from each pleural cavity and from the abdomen. The specific gravity of that from the right pleural sac was 1.028, from the left pleural cavity 1.022 and from the abdomen 1.020, while the albumin-percentages estimated according to Reuss's formula were respectively 7.52, 5.37, and 4.62. The specific gravity was taken with three different urinometers and all of them agreed, so that there could be no error.

Now these figures proved conclusively that the intrapleural fluid and probably also the intraperitoneal were not transudates and a part of a general dropsy, but were inflammatory in origin. It was then easy to understand that the anasarca of the lower extremities was the result of stasis and cachexia secondary to the condition in the great serous sacs.

Unfortunately, the man died the following day before time was given for a determination of the pathological condition underlying the exudates. In the light of the statements made by Miller that tuberculous pleurisies give exudates of the highest specific gravity and next to that carcinoma it may be surmised that the original condition was a tuberculosis of the serous membranes. But the point of this case is that the physical findings pointed at first to a general dropsy and hydrothorax. The fluid in the pleural sacs was not a transudate but an exudate.

I recall a case of a lady seen in 1894 who was the most water-logged, I think, I have ever seen. Her disease was mitral regurgitation of rheumatic origin with chronic proliferative mediastino-pericarditis. Her case has been narrated in my work on Diseases of the Heart. After heroic treatment had removed the general dropsy there still remained signs of fluid in the right pleural cavity which fluid could not be made to absorb. It was removed, therefore, by aspiration and did not recur. In her case I believe this was an exudate and that it may have been the condition which started the loss of compensation which, in its turn, brought about the general anasarca. At all events I believe such a state of affairs is not uncommon.

Before concluding this portion of the subject it should be stated that analysis of transudates and exudates shows that the amount of salts and extractives remains about constant when these two conditions occur in the same individual (Miller). On the other hand, the percentage of albumin differs and is determined by a number of factors among which is the permeability of the capillary walls. The percentage may vary somewhat in the different serous cavities in the same individual. It is greatest in the pleural fluid, next in the pericardial, next in the peritoneal and last in the subcutaneous. The percentage of albumin depends also upon the richness of the blood in albumin and, in the case of exudates, is dependent also upon the degree of irritation causing the exudate.

Various methods are in use for the estimation of the albumin; but Miller's investigations have shown that the simplest and most accurate is that devised by Reuss. This formula is as follows: Albumin $= \frac{3}{8}$ (sp. gr., 1.000) $- 2.8$. In other words, divide the last two figures of the specific gravity of any fluid by the fraction $\frac{3}{8}$ and from the result subtract 2.8.

Prognosis.—This is practically that of the primary affection. A hydrothorax, especially when double-sided, seriously endangers the individual's chances of recovery. Like dropsy in general it is to be regarded as a sign of the gravity of the original disease rather than a sign of grave danger in itself.

Treatment.—This is in reality the treatment of the condition responsible for the hydrothorax and does not need to be given here. However, we occasionally see cases in which we are called on to decide whether or not to remove the fluid in the pleural cavity. If it is occasioning serious pressure it should be withdrawn the same as if it were an exudate. It may recur, but before it does, time is afforded for the trial of remedies intended to maintain free outflow of serum from the intestines and to invigorate circulation and to improve nutrition. The hydrothorax as well as the general dropsy is often wonderfully removed by incision of the œdematous ankles under strict aseptic precautions, or by the insertion of Southey's tubes into the loose subcutaneous areolar tissue of the ankles.

II. HÆMOTHORAX

Ætiology.—By this term is meant an escape of blood into the pleural sac. It is to be distinguished, therefore, from hæmorrhagic effusions or serous exudates tinged with blood. Hæmothorax is the result usually of injury to or rupture of some vessel, as an internal mammary or intercostal artery or the aorta. Perforation of an artery traversing the walls of the chest may result from a penetrating wound by bullet or cutting weapon and may prove a very serious affair. It is possible also for a careless or inexperienced surgeon to wound the artery in the operation of thoracentesis. Fowler mentions the very instructive instance of fatal hæmorrhage into the pleural sac from an aneurysm of the internal mammary artery after an operation for empyema.

Fractures of ribs or crushing injuries to the thorax, may occasion hæmothorax through laceration of an intercostal artery or of the aorta. When the smaller vessel is ruptured the hæmorrhage is not likely to be great; when, on the contrary, the aorta is ruptured the amount of blood that escapes is large and death ensues. The bursting of an aneurysm of the ascending portion of the arch is very likely to cause fatal hæmorrhage into the right pleural cavity.

An endothelioma of the pleura (Fraenkel) is sometimes responsible for escape of dark venous blood into the sac. This is different, of course, from the bloody effusion that sometimes is observed in cancer of the lung. Certain cachectic or blood states are also responsible for hæmothorax. Thus it may be observed in the course of severe scorbutus, as in some of the provinces of Russia.

Morbid Anatomy.—The changes observed are those of the traumatic or pathological condition that has led to the hæmorrhage. The blood itself may be fluid if an autopsy be made soon after the accident. In other cases the blood is likely to be coagulated. The lung is compressed, i. e., collapsed, and there is more or less displacement of the heart depending on the amount of blood present. Death is not the invariable result, for coagulation of the blood may take place and the fluid portion be absorbed and, in the course of time, even the solid constituents. Such is likely, however, only in small hæmorrhages.

Symptoms.—These are determined by the amount of blood effused. If it be small there may be no other effects than such as would attend the collection of an equal amount of serum in the pleural cavity. There are also the physical signs of fluid accumulation in the sac. Ordinarily, however, there are the symptoms of hæmorrhage, faintness, weakness, pallor, etc., and the individual experiences more or less dyspnœa and rapidity of the heart's action. In time these symptoms wear away, but if the hæmorrhage chance to come from an aneurysm, death is likely to result either quite speedily or after a few hours.

Physical Signs.—These are essentially the same as in serous accumulation within the pleural cavity. There are dullness extending from the base of the chest upward, diminution or abolishment of the pectoral fremitus, feebleness of the voice-sounds and diminished intensity or absence of respiratory murmur over the area of dullness. The upper line of dullness may shift with change of posture but fails as soon as coagulation has occurred.

Diagnosis.—If the hæmorrhage be small it may be readily overlooked. A correct diagnosis is possible if symptoms indicative of hæmorrhage occur, e. g., in a person who has received an injury or is known to be the subject of an aortic aneurysm, and if, at the same time, signs in the chest be discovered pointing to fluid collection in the pleural cavity.

An exploratory puncture will probably determine the nature of the case; and yet one should remember that pure blood may be obtained when the sac is obliterated by an organized membrane. In a case of this kind, however, the history and symptoms are not those of a suddenly developing fluid in the chest.

Prognosis.—This is grave unless the bleeding vessel can be reached by the surgeon and ligated. Consequently, when hæmothorax comes from rupture of an aneurysm death is likely to be the ultimate issue. Injuries to the chest-wall are generally amenable to surgical treatment.

Treatment.—This is surgical, as far as it is possible to do anything. In the case of a wound of the chest it is probable that the hæmothorax is due to injury of an intercostal artery. In such a case, therefore, the surgeon should interfere without delay even if he have to resect in order to reach the bleeding vessel. If it be concluded from the physical signs that an extensive clot has formed it may be well to open the pleural cavity and empty it of the blood-clots. In the event of the rupture of an aneurysm no treatment will be of avail.

If the hæmorrhage does not prove speedily fatal and surgical aid is not at hand appropriate symptomatic treatment should be instituted in the hope of tiding the patient over the present peril and of arresting the hæmorrhage so that absorption may ultimately take place.

III. PNEUMOTHORAX

This term, which was introduced into medicine by Itard in 1803, signifies an accumulation of air or gas in the pleural cavity. Although cases are occasionally encountered in which air alone is present, still in most instances fluid of some kind also exists. Accordingly, other adjectives are prefixed to designate such a combination. Thus, we have hydropneumothorax when serous exudation and air are both present; pyopneumothorax when pus as well as air is found, and hæmopneumothorax when blood is associated with the air.

As the clinical picture produced by these various combinations does not vary essentially in each instance and as these several conditions may be regarded as modifications of pneumothorax they will be considered under the caption given to this chapter.

One occasionally sees the terms pneumohydrothorax and pneumopyothorax. They also signify the association of air with serum or pus, as the case may be, and were introduced for the purpose of distinguishing those cases in which the collection of air was the first step and the accumulation of fluid the second step in the morbid process. Although theoretically excellent, these terms are practically unimportant because of the fact pointed out by Rosenbach that clinically it is not always possible to determine which collected first in the pleural sac, the air or the fluid; neither does the exact sequence of events have any bearing on treatment.

Ætiology.—Two great groups of cases of pneumothorax must be distinguished, namely, the secondary and the idiopathic or spontaneous. The former is the one usually met with and is the more serious. It is the one that forms the subject of this chapter, although a short account of spontaneous pneumothorax will be given before the subject of ætiology is concluded.

In discussing the ætiology of the secondary cases we may again subdivide them into (1) those in which the pneumothorax arises from loss of continuity in the pulmonary parenchyma and (2) those in which air or gas gains access to the pleural cavity from outside the lungs.

(1) **Pulmonary Tuberculosis.**—This is by far the most frequent predisposing cause of pneumothorax, although the actual number of tuberculous cases thus complicated is small as compared with the number of cases of pneumothorax attributable to tuberculosis. Thus, writers are quite generally agreed in the statement that tuberculous disease of the lung is the underlying cause in about 90 per cent of the instances of pneumothorax. On the other hand, out of 58,731 cases of pulmonary tuberculosis only 433, or less than 1 per cent, developed pneumothorax (Fussell and Riesman). Among 3,415 cases of tuberculous disease Galliard found 36 of pneumothorax, or 1.054 per cent, whereas of 1,000 cases of lung tuberculosis at Brompton Hospital there were 55 of pneumothorax, or 6.5 per cent. Accordingly, the occurrence of this complication may be said to range between 1 per cent and 6.5 per cent.

The mishap may take place in either acute or chronic phthisis, and it results from necrosis and rupture of the pulmonary pleura immediately overlying a superficially located vomica or caseous focus. The perforation may occur during some expiratory effort as coughing, sneezing, straining, etc., or it may take place independently of effort, as during sleep.

(2) **Pulmonary gangrene and abscess,** particularly the former, are accounted the next most frequent cause of the escape of air into the pleural sac. The gangrenous zone is usually situated close beneath the pleura in these instances but need not necessarily be, since the destructive process may extend until it invade the membrane. The result is a pneumopyothorax of so intense a character that death is very apt to follow.

(3) **Embolic infarct** is another but rare cause of pneumothorax and since it is a septic infarct that may so act, an exudation of pus is apt speedily to follow the escape of the air.

(4) **Acute broncho-pneumonia** is said to be an occasional cause in children (Barthez and Rilliet), whereas *acute croupous pneumonia* does not so act, except in so far as it may occasion a gangrene and thus produce necrosis and perforation of the pleura.

(5) **Emphysema** is another condition which may, under some circumstances, be responsible for a pneumothorax. The emphysema may be vesicular and a dilated alveolus may, during cough or unusual expiratory strain, give way and permit air to enter the pleural cavity; or a bleb, such as is seen in interstitial emphysema and which is usually situated along the border of the lung, may burst under the same conditions. Unverricht expresses a doubt of such a cause of pneumothorax, but cases have been reported in England which prove its occurrence.

(6) **Whooping-cough** is a disease in which the rupture of an alveolus is possible and instances are on record of the occurrence of pneumothorax during an unusually violent paroxysm of cough in this complaint. It is more common, however, for subcutaneous emphysema to take place, the experiments of Champneys having shown that when an alveolus ruptures, the air makes its way beneath the pleura to the mediastinum and thence along the deep cervical fascia to the tissues of the neck.

The degree of intra-alveolar air-pressure which a healthy lung will endure (3 to 9 inches of mercury, Hutchinson) enables one to comprehend why some clinicians should doubt the occurrence of pneumothorax as a result of laceration of the lung by powerful expiratory pressure. Yet when we consider that in pertussis the pulmonary parenchyma cannot be said to be in a state of health it seems not unreasonable that pneumothorax may occasionally result; also that a person whose lung is emphysematous is exposed to the possibility of such an occurrence, although instances of the kind are extremely rare.

(7) **Trauma.**—Instances are on record of the development of pneumothorax after a severe crushing injury of the chest, even without fracture of the ribs. There are two recorded cases in which a main bronchus was torn in two and air escaped into the pleural cavity. But without so grave an accident as this the parenchyma may suffer a laceration with consequent pneumothorax. Exceptionally this accident has followed unusual physical exertion, but the injury and its consequences are usually recovered from.

It is possible also for a penetrating wound of the chest-wall and lung to permit the entrance of air into the pleural cavity, but fortunately this result is extremely exceptional. The experience of military surgeons is that pneumothorax from bullet wounds, even when the missile passes clear through the thorax, is far less likely than is extrusion of the lung-tissue from the wound. On the other hand, laceration of the pulmonary pleura by a fractured rib may be a cause of pneumothorax, Turner having found 4 instances out of 247 cases.

(8) **Echinococcus cyst** of the lung is another though very rare cause of air in the pleural cavity. The pneumothorax can only occur when the cyst bursts into the pleural sac while at the same time communicating with an air-tube or lacerating the surrounding lung-tissue.

(9) **Empyema.**—The rupture and evacuation of an encysted pyothorax into a bronchus would *a priori* seem to furnish a condition favorable to the development of pneumothorax. As a matter of experience this is exceedingly rare and some authors doubt the possibility of its occurrence. The vent through which the pus escapes into the lung is so small and the pressure of the discharging exudate is such that air cannot make its way past into the pleural cavity.

The entrance of air into the pleural sac from outside the lungs is very rare and the predisposing causes are not very numerous. Penetrating wounds to the chest-wall would appear theoretically to be capable of producing pneumothorax, but, as a matter of fact, most of them do not permit the ingress of atmospheric air to the sac. Surgical operations on the chest-wall do, on the contrary, result sometimes in the entrance of air into this cavity; but the resulting pneumothorax does not form a clinical entity.

(10) **Perforation of the pleural sac by various abdominal lesions,** e. g., by gastric ulcer, has been known to occur. It is so uncommon an event, however, that as compared with the other causes it may be almost ignored. Nevertheless, the possibility of such an occurrence should never be forgotten.

The **spontaneous evolution of gas** within the pleural cavity is a subject that has given rise to much discussion and speculation. It was believed at one time that when a collection of fluid in the sac was followed by the generation of gas without rupture of the pulmonary parenchyma or any other discoverable means of ingress of the air, the pneumothorax developed in consequence of chemical decomposition of the fluid.

Several explanations of this as yet not fully understood pneumothorax may be advanced. It is possible that the air may enter through a loss of continuity in the pulmonary parenchyma and pleura, so minute that it escapes detection; or that the wound may become healed and leave no traces of its existence. It is not impossible that some minute histological change has taken place in the tissue of the lung in consequence of which air is enabled to make its way by degrees into the pleural cavity. Thirdly, it is possible and indeed very probable that neither of these two hypotheses is correct but that the pneumothorax is the result of the action *in situ* of gas-forming bacilli.

The formation of gas in other parts of the body and in other serous cavities is now so well known that, it seems to me, the only position tenable with reference to pneumothorax formed without solution of continuity is the one of the action of micro-organisms capable of generating gas.

Idiopathic or Spontaneous Pneumothorax.—These terms are employed to designate a set of cases in which no underlying pathological condition can be discovered at the bedside to account for the sudden accumulation of air within the pleural cavity. They are believed, therefore, to arise independently of associated pulmonary disease. Fussell and Riessman reported two instances of what they believed to be examples of spontaneous pneumothorax and in their paper gave 56 as the total number of cases they had been able to collect from the literature up to that time—1902. Cases of this kind do not come to autopsy and hence the assumption of their idiopathic origin rests on the failure of the observers to detect signs of some predisposing affection and on the patient's recovery with subsequent good health.

Thus, one of Fussell and Riessman's cases was in a girl of twenty-one whose previous health had been excellent and in whose family or personal history there was nothing to suggest the possibility of pulmonary tuberculosis. This girl was seized suddenly, while asleep, with pain in the side and rapidly supervening dyspnœa. Upon the doctor's arrival early next morning he discovered signs of left-sided pneumothorax. Fluid did not develop and at the end of two weeks the young woman was able to resume her ordinary duties. She was in the possession of good health at the time of the report, seven years following. The other patient was a man of twenty-seven, a teamster, who, for a month prior to admission to the hospital, had had a cough which he attributed to a cold from exposure. The night immediately preceding his admission his cough was especially severe and he was seized with a sharp, stabbing pain in the right side. Examination disclosed a right-sided pneumothorax also without fluid. He was well in a week's time.

In both these cases the observers were unable by the most diligent physical examination to discover signs of any associated pulmonary disease. Indeed, the female patient had been carefully examined by Fussell only a week prior to her illness with wholly negative results. Without wishing to cast doubt on the accuracy of the examinations in these two cases, I yet am strongly inclined to doubt the absence of some underlying lesion. As previously stated such a great degree of intra-alveolar pressure is required (3 to 9 inches of mercury) to rupture a healthy lung that it seems incredible for rupture to take place during the quiet respiration of sleep without some predisposing disease. Moreover, in the case of the teamster there was a history of antecedent cough; and without a tuberculin test, an X-ray picture or an autopsy no one can assert without the possibility of contradiction that his lung was perfectly healthy.

It seems to me, therefore, that reasonable doubt may be thrown on the idiopathic nature of such cases. Obvious signs of some predisposing disease are not present, but our imperfect methods of examination do not preclude the possible existence of disease.

As regards the ætiology of these spontaneous cases nothing can be said further than that in the majority of instances the accident occurs during some unusual exertion as coughing, lifting, playing some violent game, etc. In some of the instances, therefore, it may be granted that physical effort, e. g., a game of football, is sufficient to occasion rupture of a lung, if, for instance, a squeezing injury of the chest is experienced; but it is rather difficult to understand why, under such circumstances, the injury should not result in more permanent or serious mischief. Nevertheless, in our present

gnorance we are forced to accept such facts as we can observe and the
est content.

It is interesting to note the ages at which these cases of spont
neumothorax have occurred. According to Fussell and Riessman th
as follows: of the 48 cases in which the age was given 1 was belo
13 were between ten and twenty; 30 were between twenty and forty;
over forty, while the remaining 8 whose exact ages were not stated w
young adults.

As respects the symptoms and diagnosis of these cases there is n
to distinguish them from the secondary form of pneumothorax exce
fact that fluid very rarely becomes added to the air in the pleural sac.
prognosis is good as all of the cases regain health. The only tre
required is rest in bed and protection from further injury or strain.

Morbid Anatomy.—The changes discovered in making a *post-m*
examination in a case of pneumothorax present nothing at all charac
aside from the perforation or fistula through which the air escaped.
unless great care is exercised in opening the thoracic cavity, positive ev
of the presence of air in the pleural sac may be missed. Either one of
procedures is recommended for the positive detection of a pneumot
The external tissues may be carefully dissected away so as to uncov
parietal pleura over the supposed seat of the pneumothorax. Then,
the membrane be thickened or covered with fibrin, the anatomist ca
through it and perceive whether the visceral pleura lies in contact wi
costal layer or is separated a little distance from it. In case this m
of examination is unsatisfactory, the pathologist may arrange the i
ment in such a manner as to form a pouch into which water is p
Then the pleura is punctured underneath the water. If air is pres
the sac it will escape in the form of air-bubbles while the water is ru
into the cavity. If preferred the abdominal cavity may be filled with
and the pleural sac be pierced through the diaphragm, and the esca
air be noted. The audible escape of the pent-up air can take plac
when the air is confined under positive pressure, which is not usual.

After the thorax has been opened, the lung, heart and pleura will
found to present very much the same appearances as in cases of pl
with a sero-fibrinous or purulent exudate. The lung is more or less coll
the heart and mediastinum are displaced toward the opposite side an
pleura may display signs of recent inflammation; or, in exceptional ca
may look comparatively healthy.

In most cases a perforation in the visceral pleura is readily disco
though in some no opening can be detected because the tear has b
sealed or is covered over by lymph. If a fistula exists it may be an
one, or it may be of the kind known as valvular, i. e., a flap may exist
during expiration permits closure of the opening; hence the term *closed*
mothorax is applicable to cases in which no fistula is discoverable and
pneumothorax to cases in which a permanent fistula is present.

The *shape of the perforation* is usually circular or nearly so a
edges may be smooth and regular in cases of considerable standing or i

lar and thickened when the condition is a recent one. The *size* varies considerably but in general is small, less than ¼ inch (West), and yet it is stated that openings as large as a silver quarter, i. e., about an inch in diameter, have been occasionally discovered.

The site of the fistula is not always the same. It is most often situated at the lateral and posterior aspect of the lung although the anterior surface is not an uncommon site. West states that its situation is most frequently in the lower portion of the upper lobe and attributes this fact to the presence of adhesions above, since the perforation is apt to occur just below the level of adhesions. The fistula may be found, however, in the middle lobe or in the upper portion of the lower lobe.

Whatever be the seat of the opening it is usually situated over a cavity which is superficially located and is of recent formation, for the walls of old vomicæ are apt to be fibrous and hence not favorable to rupture. Either lung may be the organ affected and apparently in about equal proportion. At all events figures show but insignificant differences in the liability of the two sides to be the seat of pneumothorax. If any preponderance exists it is in favor of the left, as the more frequent seat of this affection.

Mechanism of the Production of Pneumothorax.—Experiments on living animals show that the separation of the two pleural surfaces by the introduction of either fluid or air through an opening in the chest-wall is a matter of great difficulty. This is owing to some special provision which maintains the two membranes in accurate coaptation. This provision is to be found probably in the tissue-tonus of both the lung and the chest-wall by which the natural tendency of the organ to elastic recoil is counteracted, in other words, in the expansibility of the lung.

When, because of disease, e. g., pulmonary tuberculosis, the parenchyma has become rigid, its normal expansibility is lost. If a perforation occurs in its enveloping membrane it is a comparatively easy matter for the air contained in the bronchi communicating with the fistula to pass into the pleural sac and separate the two serous surfaces.

If the surrounding lung substance is healthy, the lung can expand and prevent the formation of an extensive pneumothorax. But ordinarily such is not the case; the pressure of the air in the fistulous tract is greater than that in other parts of the lung and air passes into the pleural cavity. If the walls of the fistulous communication between the bronchi and pleural sac are rigid and cannot collapse during expiration, then air may enter the pleural cavity during both respiratory acts. Clinical proof of this is found in the circumstance that over the site of the perforation both inspiration and expiration have an amphoric quality.

It is generally held that the condition favoring positive pressure within the pleural cavity is a valvular slit of such a kind that it opens to admit air during inspiration and closes so as to prevent exit of air during expiration. Rosenbach holds that expiratory power is too feeble to force out the air during this phase of respiration, even when the fistula is freely open, since the air in thus passing out would have to meet and overcome the column of air in the bronchi. *The conditions that raise the pressure in* *the sac to a positive degree are the elasticity of the chest-walls, the inability on the part of the lung to expand and the retraction of the neighboring viscera in consequence of the loss of normal tonus on the part of the lung.* Under these circumstances the degree of pressure within the pleural cavity may become extremely distressing and even dangerous.

Symptoms.—The establishment of a pneumothorax may be declared by very abrupt and characteristic phenomena or it may take place so insidiously that its presence is not suspected until distinctive physical signs have become established. In the former and more usual class of cases the individual suddenly experiences a sharp pain in the side, oftentimes with a sensation that something has given way in the corresponding half of the chest. The symptoms of shock now rapidly supervene.

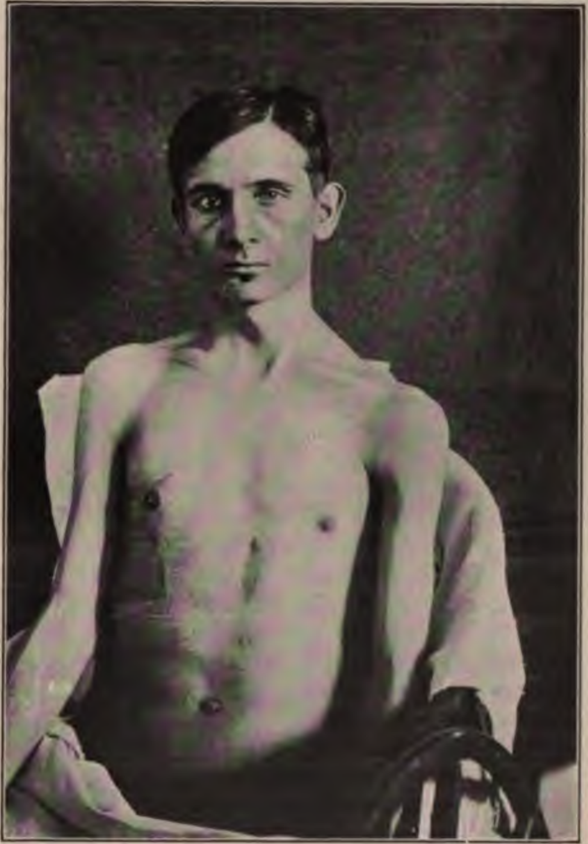


FIG. 104.—Inequality of two sides of chest in pneumothorax.

The countenance assumes a pale, anxious appearance; the action of the heart grows rapid and feeble so that the pulse may be scarcely perceptible. The temperature falls and the surface of the body grows cold and bedewed with perspiration. The breathing becomes hastened and dyspnœa may develop. Before long the patient may be obliged to sit up to breathe, or to lie on the affected side to afford the sound lung all the room possible for the performance of its own function as well as that of its disabled fellow. If the positive air-pressure within the pleural cavity grows extreme, the sufferer may manifest evidence of imminent suffocation and call feebly for help.

Under such circumstances the face becomes cyanosed and inspection of the chest shows a marked contrast in the appearance of the two halves (Fig. 104). One side is seen to be distended and nearly or quite motionless, while the other works with abnormal rapidity and vigor. If the distended half is palpated the intercostal spaces are found obliterated and on percussion the note is sonorous and drumlike; or *if the distention has reached an extreme grade the note may be dull* and the resistance be increased; a condition which suggests accumulation of fluid instead of air in the pleural sac, and which I have known physicians to find extremely puzzling.

Pain.—Not all cases display uniformity as regards this symptom. It occasionally happens that the advent of the pneumothorax is not announced by decided pain and it is in such cases that the affection may be said to be insidious in its onset. When pain is experienced it is usually severe, and that such should be the case is not strange when we consider the great sensitiveness of the pleura. The pain is caused by the sudden laceration of the membrane or by the tearing asunder of adhesions, while it is very possible that the resulting and often great distention of the thorax may also be an element in the production of this symptom.

Cough.—This likewise may or may not be a distressing symptom. Ordinarily it is not a marked feature, but now and then cases are seen in which cough is very frequent and very distressing. When it occurs it is quite possible that it contributes to the establishment of high intrapleural air-pressure.

Dyspnœa.—Few if any cases of complete pneumothorax are seen in which difficulty of breathing does not make itself felt. It may be said that, as a rule, the degree of dyspnœa is proportionate to the rapidity with which the air distends the pleural cavity and to the degree of pressure. The breathing is accelerated in all cases, therefore, while in some it may be extremely hurried and extremely shallow. Indeed, in some instances the dyspnœa attains a grade of intensity seldom seen in any other affection.

The **pulse** is, as previously stated, much accelerated and in some instances so weak as to be thready and almost imperceptible. I have known feebleness of the heart action to be so conspicuous a feature as to mislead an experienced physician into the supposition that the dyspnœa was the expression of cardiac asystolism instead of both phenomena being symptomatic of a common cause.

Temperature.—In the commencement of the difficulty this is lowered in consequence of shock, but after a time reaction sets in and the temperature becomes febrile. Such is certainly the case in pulmonary tuberculosis, and when the pleura becomes inflamed with the formation of a purulent exudate (pyopneumothorax). The degree of the pyrexia differs, and there is nothing peculiar or characteristic in its type.

Prostration.—This usually becomes well marked and in all the cases I have seen the sufferers have displayed very noticeable weakness and disinclination to exertion. Since nearly all patients with pneumothorax are consumptive, it is not unreasonable to assume that the primary disease has much to do with the asthenia.

The conversion of a pneumothorax into a hydro- or pyopneumothorax by the development of an exudate is very frequent, as already stated. It does not materially change the clinical picture except in so far as it alters the physical signs. This it does in quite a striking manner as will be set forth when considering the physical findings.

Partial pneumothorax is one in which adhesions confine the air to a circumscribed portion of the pleural cavity. Any part of the sac may thus be the seat of the air-accumulation and, if it be small or irregular in outline, the pneumothorax may be most difficult if not impossible of detection.

The symptomatology of the disease under consideration is well shown by the two following cases of which only the essential features are given. Both were admitted to Ward 4, Cook County Hospital, in the Fall of 1902 and proved all the more instructive because of their points of contrast as well as similarity. B. L., a Russian, laborer, age thirty-five, admitted December 6th and discharged two days later, was so short of breath and found talking so difficult that he gave a very meager account of his illness. It was ascertained, however, that he had been troubled by cough somewhat intermittently for two years. About two weeks prior to admission he had been seized with a paroxysm of coughing and difficulty of breathing which he thought was the result of having taken cold. Since that attack he had felt very weak and suffered from a dull pain low down on the right side of the chest.

On examination it was found that he could not lie on his back because of his dyspnœa. He was breathing 56 times a minute; his pulse was 112 and his temperature was 100.8° F.; his expression was that of one in great distress, and emaciation was marked. The skin of the chest was pale and moist; the infraclavicular regions were sunken; pectoral muscles were atrophied; respiratory movements were limited; apex-beat was visible in the sixth left intercostal space one inch outside the nipple line; there was bulging of the right side extending from the second to the fifth interspace in front and from the posterior axillary line to the mammillary line. Palpation—pectoral fremitus was well marked over the left apex down to the second rib, and there were occasional râles and what seemed to be a pleural rub over both lungs, except from the eighth to the twelfth rib on the right side corresponding to the prominent area already mentioned. Percussion—left apex, anteriorly, impaired above clavicle and to second rib, normal resonance below; posteriorly, dull to level of second dorsal vertebra, resonant below; right lung, anteriorly, fair resonance to the sixth interspace; posteriorly, fair resonance to level of third dorsal vertebra, and dullness from the third to the eighth vertebra and from the eighth to the twelfth dorsal spine hyperresonance of tympanitic quality, extending forward to the anterior axillary line; over this area of exaggerated resonance the percussion-note was metallic and the coin sound could be elicited. Auscultation—left lung, anteriorly, harsh breathing over upper portion and exaggerated breathing below; posteriorly, bronchial breathing over the dull area at apex and vesicular breathing below, with occasional pleuritic friction; right lung, anteriorly, breath-sounds seem nearly normal; posteriorly, respiratory murmur diminished and distant over the dull area from third to eighth dorsal spines, and over the region of loud drumlike resonance amphoric breathing and the succussion splash.

In this case the physical findings indicated tuberculous disease of the left lung and a right-sided pneumothorax with a small amount of fluid. The right lung was collapsed and probably tuberculous and, judging from the amphoric breathing at the right inferior back, the perforation was on the postero-lateral aspect, where, according to West, it most often takes place. The heart was dislocated to the left and downward. The dyspnœa was very great and seemed out of proportion to the distention of the thorax.

In the following case the onset was abrupt and stormy but the course was prolonged and the subjective symptoms were not especially severe. In that respect it presented a contrast to the one preceding. J. H., Irish, laborer, aged forty-three years, was admitted to Cook County Hospital October 9, 1902, and was discharged November 28, 1902, not improved. The previous history was indefinite as regards symptoms of pulmonary disease. He did not admit chronic cough or loss of strength, but said he had lost twelve pounds in weight in about a year. He thought he had taken cold about a week before the onset of his present trouble, from having worked in a windy basement. On the morning of October 8th, the day preceding his admission, he arose feeling well and that day superintended a small gang of laborers. Early in the evening he rolled himself up in his blanket and lay down on his cot in a tent. Very soon thereafter he began to cough and coughed violently for five hours, when he sought the hospital and was admitted at 12.40 A.M.

He was able to walk into the ward but was breathing 30 times per minute; his pulse was 100 and his temperature was 99.8° F. On examination he was seen to be a well-built man in a fair state of flesh and nutrition; the respiration was hurried but not laborious and he did not appear in distress; the left half of the chest was perceptibly larger and rounder than the right and on more careful inspection it was seen that the movements of the left side were abolished, respiration being apparently performed by the right lung alone; the apex-beat was not visible in its usual situation, but there was an impulse in the fifth right interspace about 1½ inches from the sternum. On palpation, pectoral fremitus was everywhere present on the right and everywhere absent on the left, and on this side the intercostal spaces were smoothed out. Percussion showed relative dullness over the entire right chest, and on the left hyperresonance from apex nearly to base, the note being loud and drumlike; in the dorsal decubitus there was flatness posterior to the mid-axillary line, and upon the patient's sitting up the tympanitic note at the front base became replaced by flatness which reached to the level of the fifth costal cartilage nipple line; over the area of hyperresonance above the level of the fluid a coin sound could be heard with great clearness and intensity. On auscultation the breath-sounds at the right were harsh and broncho-vesicular, while on the left they were diminished above and wholly absent below the fourth rib in front; on shaking the patient a beautiful splashing sound was elicited; after speaking, a metallic tinkle or gurgle could be heard; the heart-sounds were audible at right of sternum but not in their normal situation.

During the next few weeks the level of the fluid slowly crept upward to the second rib in front, and the heart became more displaced to the right.

The general condition did not appear to grow worse, however, and dyspnœa was never a marked feature. Tubercle bacilli were repeatedly found in the sputum. Exploratory puncture showed serous fluid, and positive intrapleural pressure was demonstrated. The temperature was never high and strength did not seem diminished during his stay in the hospital.

Physical Signs. Inspection.—The phenomena noted by inspection are commensurate with the degree of intrapleural pressure the same as in pleurisy with effusion. Indeed, they are so similar in these two affections that the description of one may answer for that of the other. The patient may display orthopnœa of an intense grade or may be seen lying on one side. This is usually the side on which the pneumothorax is located, but I have known the individual after his disease had lasted for a week or so, to lie on the opposite side.

The respirations are hurried and difficult and if pressure is very great the face is cyanotic and anxious and the superficial cervical veins may be turgid and pulsating. Upon uncovering the chest this is seen to present more or less inequality as regards the contour and respiratory movements of the two halves.

The side affected is visibly larger and more rotund while the natural depressions corresponding to the intercostal spaces are obliterated. There is, moreover, a complete or nearly complete loss of motion on this side, while its fellow moves with unwonted rapidity and force. If the apex-beat is visible it is seen displaced toward the opposite side and there may be noticeable pulsation in the epigastric region. The downward dislocation of the liver is shown by fullness in that region and in some instances its lower border may be shown by a perceptible line of depression extending across the abdomen. The more emaciated the patient, the more apparent are these various peculiarities.

Palpation.—The resistance of the distended half of the chest is increased and vocal fremitus is wholly lacking or very feeble. If adhesions confine the air to the lower segment or to some other circumscribed portion of the chest, pectoral fremitus is absent in this area and preserved in other parts. Palpation also enables one to appreciate the displacement of the heart and of the abdominal organs. The hand passed over the side perceives the smoothness and increased tension of the intercostal spaces.

Percussion.—One of the most striking characteristics is the intensity and low-pitched resonance of the percussion-note. This may be so full and drumlike that the note over the sound lung may seem dull in contrast. In complete pneumothorax, moreover, the hyperresonance is found to occupy the entire half of the thorax and, indeed, to extend beyond the normal limits of pulmonary resonance. It reaches across the median line and encroaches upon or obliterates the area of cardiac dullness, while inferiorly the hyperresonance, in extreme instances, may reach as far as the lower costal margin, the liver-dullness being found entirely below the arch of the ribs.

This is not the only peculiarity displayed by the percussion-note, however; for if the ear or stethoscope be placed against the chest while percussion is made at some nearly opposite point by means of two hard substances, e. g., two coins, the note is generally heard as a clear silvery tone (the coin or bell sound). This tone is due to the production of overtones and for its production requires certain conditions of tension, and hence it is not always present in perfection owing to the fact that the air is confined under too great tension. Neither is this metallic note perceptible during all stages of the pneumothorax. In some cases it develops only after the pneumothorax has existed for a time, while in others it does not appear until fluid begins to collect.

It is plain, therefore, that certain conditions are required for the production of this metallic quality of the percussion-note. These requirements are a certain degree of tension and some condition of the inclosing walls (smoothness and regularity) which favor the production of unharmonious higher tones. In my experience this bell sound is not always heard in a given case with equal clearness and perfection over all parts of the side. Why this is I cannot say unless, perchance, the existence of adhesions in some areas interferes with the necessary development of these higher tones. Certain it is that in partial or circumscribed pneumothorax this metallic sound is elicited only over the region occupied by the collection of air.

In hydro- or pyopneumothorax percussion brings out another striking peculiarity of the note, namely, its changeability in accordance with posture. In the erect position dullness is found at the base of the chest and hyperresonance above, whereas when the patient occupies the dorsal decubitus the loud drumlike resonant note extends to the lowermost borders of the thorax. This mobility of the area of dullness is the same as in pleurisy with effusion, but is far more conspicuous in the condition now described.

An exceptional percussion finding is a change in the pitch of the percussion-note depending upon the patient's position. This *Biermer's change of pitch,* as it is called, after the one who first described it, consists in a lowering of the note in the erect posture and a rise in its pitch in the recumbent position. It can be demonstrated only when fluid as well as air is present, so that the gravitation of the liquid shortens or lengthens the air-waves and thus raises or deepens the pitch of the note. It is also stated that when a large bronchus communicates with the pleural sac by means of a vomica and free opening, the percussion-note may be raised during inspiration.

Finally, attention must again be drawn to the fact that if the chest-walls be very tense and inelastic from excessive intrapleural air-pressure the percussion-note may be *dull instead of hyperresonant.* Under such circumstances it may be very easy to misinterpret the condition present unless due attention be paid to the history, subjective symptoms and other physical signs.

Auscultation.—The breath-sounds are very feeble or wholly inaudible, the latter being the case only when a complete pneumothorax has caused collapse of the entire lung. Inasmuch as the organ is rendered incapable of complete atelectasis by reason of its tuberculous disease, the breath-sounds are ordinarily diminished and of an amphoric or metallic quality. If the communication between the lung and pleural sac be large, it may be possible to locate the opening by noting the point at which the amphoric breathing is the most distinct.

The *Hippocratic succussion splash* is a phenomenon for the production of which conditions are provided only when air and fluid both are present in the pleural cavity. If the examining physician places his ear against the chest on the side of the hydropneumothorax and then gives the patient's trunk a vigorous lateral shake he hears a peculiar metallic splashing noise precisely like that produced by the agitation of water when this partially fills a large bottle. It has been claimed that a succussion splash may be perceived in rare instances over a very large pulmonary cavity containing thin liquid secretion. But this possibility is so remote that it may be practically left out of consideration. Hence the Hippocratic splash above described may be held to be pathognomonic of pneumothorax, or, strictly speaking, of air and fluid together in the pleural sac.

Metallic tinkle is another auscultatory phenomenon sometimes detected in this affection. This is a clear metallic tinkling sound produced whenever a drop of pus or other fluid falls from the inner wall of the chest into the liquid below. Hence it is most likely to be perceived directly after the patient has changed his recumbent for the erect posture. It is stated that the bursting of a bubble of mucus or pus in a bronchial tube may also possess the same metallic quality. Furthermore, it is declared (Unverricht) that if the perforation in the pleura be situated below the level of the fluid, the entrance of air during inspiration may be attended with a metallic gurgle. In the single case in which Unverricht perceived this singular phenomenon, the autopsy subsequently substantiated the correctness of the explanation given above. Leube states that in one instance of circumscribed pneumothorax of the left side the heart-sounds possessed a distinct metallic timbre.

Diagnosis.—There are few diseases in which diagnosis is so readily made. The combination of physical signs is such that even without a history of sudden onset of pain in the side and dyspnœa, the collection of air in the pleural cavity can scarcely fail of recognition. Such is certainly the case when fluid and air both are present or when the condition is a complete pneumothorax. When, on the other hand, a pneumothorax is limited by adhesions or when, for this or some other reason, the distention of the side is not very apparent and the heart and liver are not appreciably displaced, the diagnosis of the condition may not be easy. If, under such circumstances, the bell sound can be elicited the nature of the case is settled.

Should a metallic quality of the percussion-note not be perceptible the diagnosis may probably be arrived at by study of the patient's general condition, i. e., the recognition of some disease as pulmonary tuberculosis which predisposes to pneumothorax, by the detection of hyperresonance over one side or over a circumscribed area, and by the diminution or absence of breath-sounds over such an area. If such a combination exists in a young person in whom emphysema is not likely, the existence of pneumothorax may be assumed.

The following case illustrates how easy it is for a superficial or careless examiner to overlook a pneumothorax, and it is narrated that it may serve as

except, perchance, it is unilateral which, in itself, is not a frequent occurrence. (*a*) In emphysema there is no history of sudden onset of pain and dyspnœa. (*b*) If enlargement of the thorax exist this is likely to be bilateral and to display the characters designated barrel-shaped. (*c*) The hyperresonance lacks the metallic quality of pneumothorax and the bell sound is never present. (*d*) There is no mobility of an area of dullness at the base of the chest as in hydropneumothorax, and none of the other peculiarities of percussion sometimes found in the latter affection. (*e*) The succussion splash is never present and there is no metallic tinkle, but, on the contrary, there are apt to be râles of chronic bronchitis at the bases. (*f*) There is no displacement of the heart to one side, or of the liver downward.

(2) *Subphrenic Abscess.*—Under some circumstances this disease may give rise to difficulty: (*a*) The history is of some abdominal disease, e. g., gastric ulcer, periappendicular abscess, etc., instead of some pulmonary complaint. (*b*) There is no enlargement of the side or, at the most, but a very inconsiderable difference in the contour of the affected half. (*c*) The heart is not displaced to any conspicuous degree, whereas the liver is displaced downward often to a considerable extent. (*d*) Absence of cough and expectoration. (*e*) But little if any dyspnœa in consequence of pressure. (*f*) Not so considerable mobility on the part of the abscess as shown by percussion, if, indeed, any difference in the percussion findings be made by change of posture. (*g*) Vesicular breathing down to the upper edge of the tympanitic resonance due to the abscess and below this line entire absence of breath-sounds, while by forced inspiration the distance to which the respiratory murmur is audible can probably be seen to descend somewhat. (*h*) Absence of the usual auscultatory phenomena found in pneumothorax.

(3) *Dislocation Upward of the Stomach.*—Such a condition, at least to the extent of simulating a left-sided pneumothorax, can only take place when the left lung is atrophied and the diaphragm is drawn upward. (*a*) The tympanitic note lacks a metallic quality and the bell sound is not perceptible. (*b*) The gurgling noises heard in the stomach do not have the distinctly metallic quality of the metallic tinkle of pneumothorax. (*c*) There is no visible distention of the side. (*d*) The heart is not so strongly, if at all, displaced to the opposite half of the chest. (*e*) If shaking of the patient's body should produce a splashing sound this lacks the peculiar metallic quality of the Hippocratic succussion splash. (*f*) The passage of the stomach-tube will probably settle the difficulty.

(4) For the recognition of a large *phthisical cavity* the reader is referred to the chapter on pulmonary tuberculosis.

(5) *An Empyema Cavity.*—(*a*) There is a history of sudden rupture of pus into a bronchus and of its evacuation through the mouth. (*b*) Signs of dyspnœa and of other pressure-effects are all very much less than in pneumothorax. (*c*) Percussion and auscultatory evidences of the collection of air and fluid in the pleural cavity are very much less distinct, especially the metallic quality of the sounds in pneumothorax.

Prognosis.—This depends largely upon the cause of the pneumothorax and is certainly influenced by a number of conditions residing in the disease itself. An idiopathic pneumothorax is likely to be recovered from under rest and proper treatment. A complete pneumothorax in the course of pulmonary tuberculosis is likely to prove fatal and, according to West, inside of four weeks in about 90 per cent of the cases. Occasionally, however, even this sort of pneumothorax may heal or may be endured for a number of years. It may be recovered from when it is circumscribed and when the perforation becomes closed. On the other hand, it may be carried for years when the communication is free and when pressure-effects are not serious. It is more likely also not to entail disastrous consequences when air alone is introduced. When fluid also collects it is apt to be purulent and a pyo-pneumothorax may be attended with severe constitutional symptoms, independent of those connected directly with the local condition. Very great intrapleural pressure may result speedily in death unless relieved by prompt treatment.

Treatment.—In the majority of cases of pneumothorax the management must be purely symptomatic. Pain must be relieved by the administration of a dose of morphine. This is preferably administered hypodermically since thereby its action is the most speedy and certain. The exhibition of this remedy has another recommendation; namely, the quieting of the cough and the lessening of the sense of dyspnœa. It also allays the alarm which is very naturally occasioned by the suddenness and intensity with which the symptoms arise.

In addition to this simple treatment the strength of the individual should be sustained and an attempt made to rally the patient from the shock by the administration of stimulants and restoratives. Ammonia, camphor, musk, whisky, strychnine and digitalis are all suitable to this end. In short, the physician must meet the indications presented in each case. He can do nothing to promote the closure and healing of the perforation but must content himself with relieving the most urgent symptoms and with sustaining the powers of life in the hope that, in favorable cases, the process may heal spontaneously.

Operative Treatment.—In those cases in which life seems threatened by the excessive pressure of the air within the pleural cavity, the physician should at once introduce a needle into the side and thus furnish the air with a channel of escape. The relief thus afforded is said to be prompt and gratifying. This simple operation may be repeated as often as the urgency of the symptoms may require. If pyothorax be added and produce grave general symptoms it may necessitate operative intervention.

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